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For use with Documents with Protective Markings up to and including [redacted]

RC 144
3132
15-11-84

COMPENSATION (COMMONWEALTH GOVERNMENT EMPLOYEES) ACT 1971
[redacted] - Formerly Technical Assistant
Department of Defence

In the matter of the claim of [redacted] for compensation in respect of nuclear radiation damage.

DETERMINATION

1. On the evidence, before me, including specialist medical opinion, I find that the condition of chronic mild myelofibrosis suffered by the said [redacted] constituted a disease due to the nature of his employment within the meaning of section 10 of the Commonwealth Employees' Compensation Act 1930, as amended.

2. NOW THEREFORE, in pursuance of the provisions of the Compensation (Commonwealth Government Employees) Act, including sub-sections 4(2) and 104(1) of that Act, I hereby determine :-

(a) the said [redacted] suffered a disease, namely, chronic mild myelofibrosis in circumstances under which the Department of Defence would have been liable to pay compensation under the Commonwealth Employees' Compensation Act 1930, as amended;

(b) the Department of Defence is therefore liable to pay compensation in relation to the disease in accordance with the provisions of the Compensation (Commonwealth Government Employees) Act 1971.

[redacted]

[redacted]

Delegate of the Commissioner
for Employees' Compensation.

31 MAR 1983

ROYAL NORTH SHORE HOSPITAL

CA:jmc
Your Ref: 82/3152

[redacted]

The Commissioner for Employees
Compensation.
P.O. Box 196,
WODEN. ACT. 2606

[redacted]

Dear Sir,

In regard to your letters of the 1st September and 3rd December, 1982, I would like to detail the case notes as I have obtained them.

[redacted] aged 43 years was referred to the Haematology outpatients clinic on the 4th August, 1982 for investigation of splenomegaly. The event which precipitated attendance at the clinic was the recent deterioration of abdominal pains which the patient had suffered intermittently for many years. However, in the past three to six weeks, the pain had been worse and on examination by [redacted] local doctor, tender splenomegaly was noted. [redacted] stated that in 1958 at the age of 17, he worked at the Maralinga atomic bomb testing sites and that he had had excessive exposure to radiation. Six months after leaving the test sites, whilst in England, the patient had a febrile illness of undetermined cause although he was told at the time that he had signs and symptoms consistent with malaria but no definitive diagnosis could be proved. In 1969 the patient was admitted to Brisbane Hospital during which time an abnormal blood count and bone marrow investigation were found. I do not have the details of these results which will be necessary for you to obtain in order to completely evaluate [redacted]. In 1977 the patient suffered paratyphoid fever and was admitted to Prince Henry Hospital where again an abnormal bone marrow was found although a specific diagnosis was not told to [redacted]. For many years, [redacted] has also complained of pains in his right foot and also of dyspnoea on minimal exertion.

Clinical examination revealed a well looking man with blood pressure 110/70, pulse 80 and regular, respiratory rate 16 per minute. Cardiorespiratory examination was normal, several small subcutaneous nodules on the trunk and limbs were noted, there was no lymphadenopathy and abdominal examination revealed a very tender xiphisternum, borderline hepatomegaly and an enlarged spleen palpable 3-4cm below the left costal margin and the spleen was tender.

Cont'd page 2.

Investigations performed were as follows:-

Blood Count. Hb 133 g/l, MCV 85, MCH 29.7, MCHC 344, Platelets 570 x 10^9/l, ESR 5mm in 1 hour, WCC 5.2 x 10^9/l, Reticulocytes 2%, Differential on the white cell count Band Forms 4%, Seg. Neuts 75%, L 15%, M 5%, E 1%.

The blood film was grossly abnormal and showed nucleated red blood cells, moderate to marked anisocytosis and poikilocytosis, and a moderate degree of polychromasia.

Bone marrow aspiration was performed and was difficult to aspirate but from the right posterior iliac crest and from the sternum. A reduced number of marrow particles were obtained and on microscopy the particles appeared dense and abnormal. Erythropoiesis was dyserythroid. Granulopoiesis was dysplastic and only occasional megakaryocytes with dysplastic features could be found. Lymphocytes were present with occasional atypical forms.

Bone marrow trephine showed an abnormal marrow with hypercellular mass of haemopoietic marrow filling the marrow space with irregular islands of fat cells between them. All three cell lines were present but megakaryocytes appeared unusually prominent, variable in size and forming clusters. A marked increase in reticulum was present and the changes were those of a myelofibrosis.

Biochemical examination was unremarkable apart from a mildly elevated creatinine of .12 and urea of 7.4mm/l.

Chest X-ray was normal.

Liver spleen scan showed a liver of normal size with even uptake and an enlarged spleen with even uptake.

A bone marrow scan with technician 99 sulphurcolloid was performed and showed minimal uptake of sulphurcolloid in the sacrum and pelvis and no uptake in the sternum with no evidence of peripheral extension into the long bones.

Interpretation was of marrow hypoplasia.

In view of the dyspnoea, respiratory function tests were performed and showed mildly abnormal alveoloarterial oxygen difference, normal gas transfer, normal lung volumes, and normal airways resistance with normal spirometry and no significant upper airways obstruction. However, an exercise test was also attempted in order to pick up early degrees of pulmonary fibrosis. This test was aborted because of severe hyperventilation causing dizziness and distress therefore making the measurements of gas transfer questionable.

Biopsy of the skin lumps was performed and showed benign lipomata.

In summary, [redacted] appears to have marrow hypoplasia with marrow fibrosis and splenomegaly which in our opinion, is enlarged due to extra medullary haemopoiesis. I have discussed [redacted] case with [redacted], staff haematologist, who has also seen [redacted] in consultation and we agree that [redacted] is a most unusual case. In attempting to give an informed opinion on a probable cause of the condition which [redacted] has, I have tried to research previous cases of radiation exposure and various articles and books as referenced below. Unfortunately although there is a lot of literature on the effects of radiation, much of this has been directed to the effects of low level ionising radiation and the effects of acute high dose radiation exposure in various reactor site accidents. Historically it would appear that [redacted] exposure was more subacute and therefore one might expect some differences in his response to the postulated excessive radiation exposure. From examination of the literature both human and animal studies and also from the knowledge obtained from patients treated therapeutically with radiation, it is known that long term effects of marrow irradiation may result in fibrosis with marrow hypoplasia. Whether or not such marrow hypoplasia if generalised would then result in the formation of extramedullary haemopoiesis such as in the spleen, I cannot answer although theoretically this is possible and in fact known to occur in some myeloproliferative disorders of the marrow. I plan to perform isotopic studies with iron 59 and by means of counting over the spleen and other organs, we can determine if in fact [redacted] has extramedullary haemopoiesis in the spleen. As I have indicated above, it would be helpful if the original reports from Brisbane and also from Prince Henry Hospital could be obtained to see if there was also marrow fibrosis or hypoplasia noted at those times. The opinion of [redacted] and myself is that [redacted] probably has radiation induced marrow fibrosis. If such is the case, then the prognosis is difficult to determine. If the patients marrow function remains at its present level and I believe it will, the patient should not come to any harm from marrow failure. The presence of splenomegaly poses some slight risk of rupture especially if the patient is subjected to any trauma and the great danger would be if the patient needed to have a splenectomy, as we believe that most of the patients marrow function, is coming from his spleen. The increased incidence of leukaemia has been reported many times in the literature following radiation exposure which is probably best documented in the survivors of the Nagasaki Hiroshima atomic bomb explosion. Although I am unable to give the likelihood of leukaemia developing in [redacted] it is our opinion that it is certainly higher than in the normal population and if such a leukaemia does develop, it is often very refractory to normal therapeutic measures. I am uncertain as to the cause of the patients respiratory complaints and it may be necessary to attempt a repeat of his respiratory function test in the future, however, I am suspicious that there may be early degrees of pulmonary fibrosis which may progress in future years.

Cont'd page 4.

It is known in animal and human studies of radiation exposure that often the end result is replacement of the affected organs by fibrotic tissue and in [redacted] case, the ultimate prognosis would depend on the degree to which this occurs in the various organs such as the lung, bone marrow and blood vessels. In short, [redacted] prognosis is that he is likely to have a shortened life span from the effects of his disease although I can be no more definite that this.

Treatment at this stage consists mainly of pain relief as required and avoidance of any situations likely to lead to traumatic injury of the spleen. Monitoring of the patient's blood count and possibly bone marrow at regular intervals say 6 to 12 monthly will be recommended to [redacted] to look for deterioration of his condition.

Please find enclosed copies of the patients medical reports and clinical notes.

Please do not hesitate to contact me again if you need further information or clarification regarding [redacted] condition.

[redacted]

[redacted]

Haematology Registrar.

Encl:

[redacted]

[redacted] Studies in the Pathology of Radiation Disease 1965
Pergammon

Medical Research Council. The Hazards to Man of Nuclear and Allied Radiations. A Second Report to the Medical Research Council. Dec 1960
Her Majesty's Stationary Office, London.

[redacted] and [redacted]
The Medical Basis for Radiation Accident Preparedness.
Elevier Nth. Holland Inc., 1980

COMPENSATION (COMMONWEALTH GOVERNMENT EMPLOYEES) ACT 1971
[redacted] - Formerly Technical Assistant
Department of Defence

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[redacted]
Delegate of the Commissioner for Employees' Compensation.

31 MAR 1983

...ive Copy
Webb
1983

TO WHOM IT MAY CONCERN

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His outlook is genial, unruffled and tolerant. He is a willing ker, particularly when he is allowed to use his own initative, regularly working over- e in a voluntary capecity. His attitude to life is that of a genuine scientist. He be recommended to undertake any research and development task in electronics, nuclear health physics under the supervision of scientific staff. It would be relatively steful to employ him on humdrum routine tasks that would rob him of interest and itiative.

[redacted]

[redacted]

[redacted]

DOCUMENT

GROUP

SEPARATOR SHEET

RC 144
3132
15-11-84

[redacted]

8 June, 1977

[redacted]

Australian Radiation Laboratory,
36 Lonsdale Street,
MELBOURNE, VIC. 3000.

[redacted]

I am writing to you hoping you may be able to help me obtain either radiation records or medical records on [redacted] who was employed with the Atomic Weapons Testing, Health Physics Branch at Maralinga for the period 25 July 1957 to 22 June 1959.

In 1968 [redacted] entered the Royal Brisbane Hospital and while there his white cell count was found to be lower than normal. The only cause the doctors could attribute this to was the work he was doing in 1957 to 1959.

In January this year, [redacted] was again hospitalized with suspected Thypoid. The doctors' initial diagnosis was supported by a low white cell count. However further tests were conducted and it was found that [redacted] was suffering from Salmonella gastroenteritis.

As the doctors are concerned about his low white cell count he has been undergoing a series of tests in Townsville including X-rays, and pathological tests to try to determine the reasons for the low white cell count.

[redacted] is of the opinion that during his work with you he was exposed to a higher level of radiation then was considered safe. This overexposure is now causing problems with his health mainly the low white cell count.

Should this be correct I feel that he may be entitled to compensation even though the accident occurred some 20 years ago.

...2/

I have written to Weapons Research Establishment for any radiation or medical records that they might have retained. However their only records were the standard Commonwealth Public Service X Ray and medical examination for appointment. This medical examination normally does not include blood tests and as such their records would show that he was suitable for employment. These examinations were conducted in April 1964 and January 1966 prior to his hospitalization in 1968. There are no records for the period 1957-1959.

Because of the nature of his work with you he is unable to inform me of any instances when he may have been overexposed to radiation and it is in this regard that I am writing to you. As it possible to obtain some form of documentary proof that he was at some time over-exposed to rddiation, which may be used in a case for compensation. I fully realize such proof may be difficult to obtain but any assistance that you could give would be appreciated.

Yours sincerely,

[redacted]

[redacted]

DEPARTMENT OF DEFENCE

WEAPONS RESEARCH ESTABLISHMENT

SALISBURY, SOUTH AUSTRALIA • BOX 2151, G.P.O., ADELAIDE, S.A. 5001 • TELEGRAMS: "WEAPONS" ADELAIDE • TELEPHONE 2599111

ther information refer to [redacted] Ext 487 In Reply Quote SP28/5/10

17 FEB 1977

RECEIVED
AUSTRALIAN INSTITUTE OF MARINE SCIENCE
23 FEB 1977

Secretary,
Australian Institute of Marine Science,
P.O. Box 1104,
TOWNSVILLE, QLD. 4810

Attention: Personnel Officer

[redacted]

I refer to your memorandum J.F.J.:D.M.B. of 2nd February, 1977.

2. [redacted] was employed as a Temporary Technical Assistant Grade 1 with this Establishment at Maralinga from 25th July, 1957 to 22nd June, 1959, when he left by way of resignation. We have no medical records in respect of that period of employment.

3. However, he was re-employed by us as a Temporary Technical Assistant Grade 2 on 6th April, 1964, at our Woomera installation. His medical and X-Ray examinations in respect of that engagement were satisfactory.

4. On 11th January, 1966, [redacted] was Permanently Appointed under the Public Service Act and was accepted as a contributor to the Superannuation Fund - indicating satisfactory medical and X-Ray standards. He subsequently tendered his resignation and from close of business on 29th March, 1967.

5. Sick Leave records maintained at this Establishment in respect of [redacted] from 1964 to 1967 show absences totalling 1 week 5 hours 45 minutes for illnesses of only a minor nature.

[redacted]

[redacted]

xxxxxx AA47165

JFJ:DMB

2 February, 1977.

Chief Administrative Officer,
Weapons Research Establishment,
G.P.O. Box 2151,
ADELAIDE. S.A. 5001

[redacted]

Reference A: Our memorandum dated 15 July, 1976.
B: Your SP28/5/10 dated 26 July, 1976.

It would be appreciated if you could forward any records either medical or radiation that you might have on the abovenamed officer for the period 1957/58. During this time [redacted] was employed with Department of Supply, Atomic Weapons Testing, Health Physics Branch, under [redacted]

The reason for this request is that in 1968 while undergoing treatment for another illness, doctors at the Royal Brisbane Hospital found that [redacted] has a lower than normal white cell count and after extensive testing considered that this condition may have been attributed to his work with the Health Physics Branch in 1957/58. Recently after being hospitalized with severe food poisoning, doctors at the Prince Henry Hospital, Sydney, found that his white blood cell count was still lower than normal.

[redacted] has requested this information for a possible compensation claim and in case he is ever hospitalized the information may be of assistance to his doctors.

[redacted]

The following information on [redacted] employment has been found in Health Physics files held by the Department of National Development and Energy.

[redacted] attended a course of lectures and demonstrations in Health Physics conducted by the Commonwealth X Ray and Radiation Laboratories in Melbourne, from 25 July to 9 August 1957, as a member of the Australian Radiation Detection Unit. (ARDU). From mid-August 1957 to October 1957 he was at Mt Clarence in South Australia which was the ARDU base for field observations of fallout, particularly along the Alice Springs road, following the atomic tests at Maralinga.

On completion of the Mt Clarence project, [redacted] joined the Australian Health Physics Team (AHPT) at Maralinga on 11 October 1957. He was initially rostered for a 6 month tour of duty at Maralinga but remained with AHPT until the end of June 1959 when he left to take up an appointment at the [redacted] During his time at Maralinga the Australian Health Physics Representative on the Range was [redacted] It is recorded that [redacted] was the sole Health Physics officer on the Range over the Christmas period 23/12/57 to 4/1/58.

The Australian Health Physics Team stationed at Maralinga provided support for the Australian Health Physics Representative who was responsible for the day-to-day supervision of radiological safety on the Range in the inter-trials periods. Duties of AHPT included :

- control of personnel entering radioactive areas, including briefing, radiation monitoring and decontamination
- maintenance of measuring equipment
- water and air sampling
- delineation of contaminated areas
- radiation surveys of test sites
- organisation of Services training courses in radiation detection.

During trials operations, radiological safety control was exercised by specialist teams from the [redacted] in these periods, the AHPT did not operate as a separate entity but rather as individuals attached to the various U.K. health physics groups.

Personnel involved in 1956 and later test programs at Maralinga were subject to radiological safety regulations approved by the U.K. authorities and the Australian Weapons Test Safety Committee. The maximum permissible doses under these regulations were based on the recommendations of the International Commission on Radiological Protection (ICRP) and were subject to the proviso that every endeavour should

-ICRP recommendations on the maximum permissible ose for irradiation of blood-forming organs, for occupational xposure, were:

- in 1955, 0.3 roentgens per week (equivalent to 15 oentgens per year)

- from 1958, 5 rems per year (equivalent to 5 roentgens r year).

A summary of film badge records shows [redacted] eceived gamma radiation totalling 4.48 roentgens over the eriod September 1957 to June 1959.

TO WHOM IT MAY CONCERN

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[redacted]

[redacted]

HEALTH PHYSICS REPRESENTATIVE

lth Physics Group,
alinga,
TH AUSTRALIA.

HARVEY · JOHNS · OWENS · ROSS

The Principles and Practice of Medicine

Eighteenth Edition

APPLETON-CENTURY-CROFTS / New York
A Publishing Division of Prentice-Hall, Inc.

DOCUMENT

GROUP

SEPARATOR SHEET

STATEMENT OF [redacted] of

P.O. 119, Innisfail, Queensland, residential address Menna Creek, (070) 65.3160

K2144
3132
15.11.84

I, [redacted] state as follows:-

I went to Maralinga in 1957 when I was eighteen (18) (born 26.5.39). In 1957 I answered an advertisment in relation to a technical position with the Department of Supply. I was trained in Melbourne for approximately two (2) weeks from 25th July, 1957 to 9th August, 1957, in Health Physics procedures. I was actually stationed at Mount Clarence base via Woomera during the actual period of all the Antler tests at Maralinga, that is the tests on 14th September, 25th September and 9th October, 1957. The test sites were about 180 miles from Mount Clarence. My job entailed trying to position myself across the fallout path based on weather predictions and then when the fallout passed over, measuring and marking on maps what I had found. Many times the changes in wind meant that we completely missed the fallout. Other times however, it resulted in our being directly under where the heaviest fallout fell. In one case I ended up with some much fallout in my hair that it was impossible to wash it so my hair had to be all cut off. One of the things that disturbed me was the fact that quite often I would find small patches of much higher

radiation than background in areas where none of the fallout from the tests we were working had gone. It was not until much later that I was able to find out that in fact it was the path where the fallout from 1956 series of test had gone. Another part of my job at Mount Clarence was the collection of sheep thyroid glands for the measure of the Iodine 131. We would collect the thyroid glands from the sheep at a time after the fall-out was supposed to have fallen. These were then sent to Maralinga by plane and were measured to see just how much Iodine 131 the sheep had eaten. At the time there was a severe drought in the area and consequently no grass so much so that many of the graziers were paying to have sheep shipped off their properties. As a consequence some of these sheep had not eaten for weeks and of course little or no Iodine 131 was found.

About one week after the antler trials had concluded I was transferred to Maralinga to the Health Physics team under a [redacted] My work involved the control of personnel entering the test area, decontamination of personnel and clothing and radiation surveys of the actual bomb sites. I ran the laundry where radio-active clothing was washed for some time at Maralinga.

The measurements I carried out at the bomb sites involved danger because there was still high

radiation levels at the site and it was important to do these as quickly as possible to reduce the exposure time.

However, we were working a lot of overtime at the time and it was not uncommon to almost triple ones normal pay. The overtime was only available when working in the forward zone however and once our film badges and other personal monitoring devices showed we had reached a certain level we were restricted to the rear-ward zone with a consequent loss of overtime. Therefore, it was common practice among the people involved including myself that as soon as you knew you had reached the allowed level you had to leave the film badges and the dosimeters in the Land Rover so they did not register. At the time this seemed to be just a big game and the overtime was terrific. Because of this practice the recorded levels of radiation measured were only a fraction of what I actually received. I recall that on one occasion my film badge was so black that the densitometer being used could not even read how much radiation I had received. I received my major radiation exposure however during the surveys of a bomb site about six (6) months after the actual explosion. To allow repeatibility in the measurements of the craters, a set of twelve lines radiated out from the actual ground zero point. Wooden marker pegs were placed at 100 foot intervals along

these lines, and the measurements were taken at these pegs. This allowed us to get a reasonably good idea of what was going on, but it suffered from the fact that the area between the radiating lines never got measured at all. I had been surveying this crater for some months, and one day when I was out by myself doing a survey of this crater, I noticed a few anomalous readings. I decided to do a bit more investigation, and found that there was a very much higher level of radiation in an area between the normally surveyed lines. The level of radiation was such that the instrument I was using could not measure it. However there was a mode in which these instruments had been found to work, that although it could not give calibrated readings, it could show relative differences. Using the instrument in this mode, I found that this high level of radiation was coming from a number of quite discrete sources, and by using my right foot to move the dirt about I was finally able to discover that the radiation was coming from some very small metallic looking particles, so by using my foot again I was able to locate about 20-30 of these and put them into one of the tobacco tins which we used for sample collection. I put this tin in the back of the Land Rover just behind me and drove back the thirty (30) odd miles to the Health Physics Labs. When I pulled up, the rest

of the staff were already outside waiting even though I had not notified them of my arrival. The reason for them being there was that as I approached, all the instruments in the lab went haywire. We were able to quickly ascertain that these pellets were much more powerful than anything we had ever previously detected. The only way we were able to measure the pellets was by leaving them outside the building, and measuring them from outside. We were soon able to establish by use of a Pearson scanning spectrometer that the pellets were Cobalt 60 with a half-life of 5.2 years. Cobalt 60 was one of the last things we expected in the normal fallout, and certainly not at this order of level. Quickly we got in touch with the [redacted] and immediately a heavy security blanket went into effect. I was interviewed by a security officer and it was impressed upon me not to speak to any-one about this at all, particularly any Australians, no matter what their position at Maralinga. A major effort was immediately started to try to collect as much of this material as possible, but there were problems because the level of radiation was so high that it was obvious that the existing Health Physics Group Personnel could not do it because by this time we were already over our limits. At this time there were a number of Australian Services personnel

who were supposed to be there on a training course in radition detection. These people could not be told what they were working with because of the security blanket, however I made up some long scoops by tying jam tins onto wooden handles, and I showed them how to use these to collect the particles. By this time, special aircraft had been flown out from the U.K. with large lead containers, and the particles were placed into these and then flown immediately back to the U.K. As the person who actually discovered the particles, and was involved in their recovery, I was under the distinct impression that the British authorities did not want the Australian Government to know anything at all about what had happened. This Cobalt accident happened in about 1958 to the best of my recollection and the Cobalt was related to the Antler series.

At the time this actually occurred I did not seem to have suffered from this radiation exposure. However at the end of June 1959 I left Maralinga to take up a position with the [redacted] in the U.K., and some months later took up another position in London. At this time I started to suffer various fever symptoms and went to different hospitals in London for tests. The doctors in each case said the symptoms were similar to those of malaria, but blood tests showed that I

was not suffering from malaria. I returned to Australia in the early 1960's and worked first in Sydney, then at Woomera, that at Brisbane. During this time I started to suffer from extreme pain in my right foot, so severe that it would keep me awake at nights, and sometimes I could not sleep for 2 or 3 nights at a time. I went to many different doctors about the problem in my foot, and many x-rays were taken but nothing wrong physically could be found. Various suggesions were made about possible gout, but again blood tests proved that it was not gout. The final pointer to the possible problem came when I was in a Brisbane Hospital for another reason completely. The hospital had a scheme of blood tests for all patients, possibly as part of a V.D. detection campaign. My tests showed up abnormal so many further tests were done. Again symptoms similar to malaria must have been found because the doctor who was doing all the tests on me actually asked, in order to rule out all possibility if perhaps I'd ever had malaria. Finally he just happened to ask if I had ever been exposed to radiation, and when I said "yes", he immediately said that could most probably be the cause. He then arranged for marrow samples to be taken from the bones in my hips, and these showed marrow damage. I had to return to the hospital every couple of months from then on so that they

could try to determine if my marrow was still detiorating or was steady. The final conclusion reached was that marrow damage had occurred, but did not seem to be getting worse. It was however conclusively proved that as a result of the marrow damage I had a permanently low blood count.

Also about this time, I started to suffer a lot from lack-of-breath. Even just walking up a flight of steps would leave me winded, even though tests done at James Cook University in Townsville showed that I had a larger than normal lung capacity. A Townsville doctor who examined me a number of times, and who obtained my whole medical history from previous doctors, finally concluded that it was probably a form of emphysema caused by the radiation.

By 1981 the radiation had not affected me to the extent where it had incapacitated me. However the side effects of it had almost resulted in my death from other causes. For example, in early 1977 I went to Bali for holidays and contacted salmonella-para typhoid. I was flown back to Australia unconcious and put into quarantine hospital. Because of the advanced state of the disease I was treated with a rather dangerous drug Chloreamphenicol. Normally this drug is only used until such time as the blood count starts to rise. (A lowered blood-count is one of the symptoms of Salmonella-para typhoid).

However, due to my now permanently low blood count, and the fact that I was unconscious and unable to tell the doctors that, I was treated with the drug for a much longer period than normal. When I finally became conscious and was able to tell the doctors about my radiation damage they ceased using it immediately, however the damage had already been done. Seven (7) years later I still suffer gastric complaints from the overdose of Chloramphenicol. This problem of complicating medical diagnosis is one of the main problems that I have had to live with in the past. Since 1981 my health has further deteriorated and I am no longer able to work full-time.

The lack of any concern by any Government Departments has been singularly unimpressive. Firstly, in trying to get some record of my employment and medical record from Maralinga had met with the utmost resistance. I had been told many times in the past that the records were 'lost'. At one stage I was able to get a letter from some Department that I had indeed worked for the Department of Supply for the period of July 1957 to June 1979, however absolutely no mention was made of the fact that it was at Maralinga. At one stage I went to the Commonwealth Compensation Department but their response was to the effect that, well if I died they might look into it.

The DC-12 Building was part of the DCRB Group of Buildings, it was associated with the Minor Trials Kittens Tests. DC-12 Building was basically a very large hot-box, a very large and very thick lead lined box with long manipulator arms that we used from outside. It had glass windows, possibly a couple of feet thick, it was a bit difficult to tell but it was very heavily shielded. This building was used by the teams who came from the U.K. for the preparation of the Minor Trial during which time, while they were out, Australians were not allowed near the building. During the period between Minor Trials it was our responsibility to maintain this building and particularly the filtering system associated with it because of the nature of the materials that they were using inside it, most probably Plutonium. There was a lot of Radon Gas given off, this was withdrawn from the hot-box and out through some filters and a large metal ducting system. The idea of this was that the Radon had a quite short half-life and by keeping the air as it was sucked through in this long series of chambers it spent enough time in there for a lot of the Radon to decay back into a solid material, one of the decay products of possibly lead or some other isotope. There were two (2) large filters which we used to filter out a lot of the solid materials, at one stage it became necessary to replace these

two (2) filters because they had gathered enough material that air flow through them was being hindered. This was measured because there was a water pressure gauge type of thing across it which essentially measured the pressure drop across it and as the filter became more and more filled with material so the pressure drop across it increased and it got to the point where the filter had to be replaced. These filters were large steel boxes with flanges at either end where they connected on to the rest of the duct work. It was necessary to use a crane to come up because they were so heavy but it was also necessary to unbolt them from the rest of the duct work. This meant that one period after unbolting and as they were being lifted out that in fact the ends of the filters were exposed to the atmosphere. People had to be there to unbolt them and that at one stage was my job. I worked with an English Chemist whose name I cannot recall at the moment, his name was [redacted] We had no proper breathing equipment available and so I had to jury-rig some equipment. For the first filter change episode I obtained a couple of small air compressors that were used in the yellow garage and these were coupled through long lengths of pressure hose to gas masks and [redacted] and I donned a gas mask each and began one of us working at either end, to unbolt the filters. During tests

beforehand it appeared that there was sufficient air being supplied by the compressors, however, after the filters had actually been unbolted and in the process of trying to manipulate them out of the major duct line, the physical effort involved meant that our breathing rate was increased quite dramatically and it became quite apparent that there was not sufficient air being delivered by the compressors and at one stage I started to breathe in and at that point my head was right level and only inches away from the input side of the filter and I started to get leaks in the gas mask because of the insufficient air and I started to breathe in the air that was in the vicinity of the mouth of the filter. Eventually we were able to get it out and the new filter bolted in. For the second filter, knowing the problems we had had with the small air compressors which were basically designed for low volume but high pressure, I decided to use something that was high volume low pressure and finally settled on vacuum cleaners so we had two (2) vacuum cleaners which were coupled on there outlet side by long large diameter plastic tubing ~~about~~ isolate about an inch and a half in diameter again to the gas masks. Preliminary testing seemed to indicate that this was much more satisfactory, that there would be an adequate supply of air, however, again when we are at that critical stage when the filter

had actually been unbolted and was half way out, I found a problem with this plastic tubing we were using, which actually came in the form of a flat strip on a roll. The plastic pipe kinked affectively cutting off my air supply completely. It became necessary for me at one stage to completely remove the gas mask in order to breathe. I attempted to point my head away from the entrance of the filter in order to reduce any radio-active materials that I might enhail. We had to operate quickly and seal the ends of the filter being removed and then wrap it up and they were later disposed of. The point of this is that it demonstrates the lack of correct equipment that was available. We were required to perform certain operations for which there was no correct equipment available and in many cases jury-rigged equipment turned out to be safety hazards.

I also recall the problems that we experienced with the disposal of radio-active wastes in the RB-DC area. Contaminated vehicles from the front line and other materials brought back for experiment became a problem. At one stage it became necessary to do something active about the accumulated waste that we had on hand. I was given the job of disposing of this and starting a graveyard. [redacted] in conjunction with Range Authorities determined an area that was to be used and Army staff dug a

large pit there for which we would be able to use to put the drums in and they would be progressively covered over as we filled up the area. I was given the job of disposing of quite a number of these materials, some of them being wastes from the DC-12 Building, where the Minor Trials Preparations went on and others were glazing and other such materials from the fordward area. The technique I adopted was to parcel these materials up into a plastic bag and use the heat sealer from DC-12 to seal the plastic bags. I would then put this plastic bag inside a large milo tin of which we had plenty. I would then take a 44 gallon drum and cut out the top of it. I would then mix up concrete and fill the 44 gallon drum to about half way and then wait for that to set, sometimes a period of 3-4 days. I would then place the milo tin in the middle of the 44 gallon drum on top of the hardened concrete and then mix up another batch of concrete and pour it over the top until the 44 gallon drum was full. At some stage later the Army people would come in with the yellow crane and take it to the graveyard where it would be deposited. My worries since have revolved around the fact that these 44 gallon drums may well have corroded. The juncture between the two layers of concrete, the top layer was poured last and the bottom layer which was poured some time several days beforehand,

this gap may well have allowed moisture from the ground to penetrate through to the milo tins, if the milo tins in turn rusted or in other ways corroded it may well be possible for some of these radio-active wastes to have been leached out into the soil. In retrospect it was a most unsatisfactory method of waste disposal that in the absence of any better knowledge or better techniques and given that it was up to my own what-have-you to come up with some method, that was the method I adopted.

DATED this day of , 1984.

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[redacted]

[redacted]

When I joined the group at Maralinga post-Antler, we were bequeathed the job of maintaining film badge records of radiation. The film badges issued were some ancient old stock much of it already past its expiry date. These were issued at the front line caravans to personnel going into the blast areas. Many of them were collected for processing, but many of them were 'lost' or souvineered. The badges were taken back to the RB labs where we developed them using some old x-ray developer. I had never developed film in my life before but after one showing was expected to be able to do it. The dark room used for developing had no fine temperature control and usually ran very cold. The method of controlling the temperature of the developing solutions was to place the beaker containing the developer into a larger container and put a mixture of hot and cold water into the outer container until the correct temperature was obtained. After developing the film badges were then read on an optical densitometer. This instrument just measured the optical density of the film in some arbitary units. These were then converted to a radiation measurement using a curve on a piece of graph paper stuck on the side of the machine. There was NO calibration for the densitomer itself. Whenever we had to change a bulb in it the procedure was to adjust a potentiometer on it until it gave a certain reading. Where the information on what this reading really was I was never able to find out, neither was I ever able to find out where the conversion graph came from. Suffice it to say that we used this system despite many changes in production batches of fim badges, changes in the chemicals used for developing, etc. without any recalibration of the equipment. The curve on the graph paper only covered a fairly narrow range of the densities measureable by the densitomer. In some cases the film badges were so dark that the optical density reading obtained was completely off the graph. The graph was already non-linear but despite this, when this problem occurred we would just guess a value and record this. This state of affairs continued until after I discovered the Cobalt 60 at Taddje. Using various other sources available we were able to estimate the strength of one of the Cobalt pellets. This was then placed in a jury-rigged arrangement of laboratory stands in one of the compounds with an array of film badges at various distances from the source. By this method we were able to relate radiation received to the optical densitomer readings and thus produce a new calibration curve.

THYROID IODINE 131 MEASUREMENTS

While I was with the L.R.D.U. group at Mt. Clarence, part of our function was to collect thyroid glands from sheep in what was thought to be the general fallout path. Several of the L.R.D.U. members had in fact had an additional 1 weeks training in Melbourne following the 2 week general course which we all undertook. At tat time in central Australia there had in fact been a drought for about 2 years in places. In some instances sheep farmers were paying to have dying sheep removed from their property so that they did not foul the few remaining sources of water. Imagine their delight when we came along and offered them $10 (5 pounds) for the thyroid glands of just on sheep. In many cases the farmer would choose the sheep closest to death anyway. The point of the matter is that the project was based on the assumption that the sheep would have been grazing on grass and thus would have ingested any Iodine 131 which may have fallen. If there is no grass to eat then no matter how much Iodine 131 may have fallen there will be no sign of it in the thyroid glands. My belief is that the project may have been dreamed up by U.K. scientists who had absolutely no idea of the Australian outback. I also believe that the only accurate readings of Iodine 131 that may have been obtained was when the fallout from one of the earlier tests went south-east towards Adelaide instead of north-east and thus fell on reasonable pastures where sheep were in fact grazing. These measurements unfortunately were not done by Maralinga scientists and were thus discounted.

THE DOSIMETERS.

Quartz fibre dosimeters were used in the forward area with the [illegible] of being able to get instantaneous readings of radiation received. [illegible] different types of instruments were in use, one with a 10 times higher range than the other. If my memory serves me correct I believe that one may have been 0-50 and the other 0-500. In the the post-test phase when radiation levels had reduced somewhat only the 0-50 instruments were supposed to be issued but because of the limited number of them available quite often 0-500 instruments were issued which would not have registered anything. The quartz fibre dosimeters in use had a frasile quartz fibre assembly and microscope optical assembly to read it with. Because of the frasile nature of them many of them were in a damaged condition e.g. either the fibre could not be seen at all, or there might be no movement of the fibre between the charged and discharged states. In many cases I found that some of the personnel working in the forward area would demand a dosimeter even though no workable ones were available because many of them were under the mistaken impression that the wearing of them prevented on from receiving any radiation. The dosimeters were electrically charged by means of a battery operated charging unit. The majority of the charging units were defunct because of different electronic faults, and it was difficult to obtain good batteries for the remainder. A large chest freezer unit was kept at the RB unit and this contained hundreds of batteries of different types. Most of the batteries were past their expiry date and had suffered water damage. The general procedure was to grab a handful of the batteries for the dosimeter chargers and hope that amongst them one could find one that still had enough charge to operate. In many cases the radiation levels recorded for persons working in the forward was 'guesstimated' based on the known levels in the general area where they were supposed to be working, and perhaps correlated with the readings of someone who might have been lucky to actually have a working dosimeter.

HOT SPOTS.

While at Mt. Clarence with the L.R.D.U. during the actual Antler trials I was in charge of a small group whose duty was to drive to the expected path of the fallout and take measurements as the fallout passed over. In some cases the wind changed and we were nowhere near where the fallout actually fell. In other cases we were directly underneath. In fact in one case I received fallout in my hair which even after about 3 washings could not be removed so the camp barber just gave me a crew cut which solved the problem. In a couple of instances while we actually in place and waiting for the fallout to arrive I decided on my own initiative to do some general background readings using a G.M. counter. Imagine my suprise when I found occasional spots where the count was already about 100 times greater that the general background. After some investigation I established that this was usually associated with clumps of spinifex, and not on the general cleared areas. My assumption was that it must have been the results of fallout from an earlier series of tests, perhaps the Buffalo series, and that wind action might have resulted in the concetrating of the actual fallout material in the vicinity of the spinifex plants. An alternative possibility occurred to me much later. It was noticed that about a year after the tests by which time there had been some rain, that when viewed from the air there was a definite increase in the vegetation in the areas where fallout had actually occurred. This was explained to me by one person who pointed out that the whole area was known to be deficient in rare earths often necessary for plant growth and that perhaps the fallout was in fact providing the necessary rare earths. Suprisingly enough this fact was bought to my attention again only about 6 years ago when I was working with a scientist whose Ph.D. thesis in botany was based in the central Australian region. In his thesis he drew attention to certain clumping of vegetation which he had observed in the area. He was not aware that atomic fallout had been over those areas and was quite suprised when I bought it to his attention. He agreed that this was the only logical explanation which satisfied his observations. This would tend to suggest then that even 20 years after the event the affect of atomic fallout in central Australia can still be observed.

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fibrosis also occurs more frequently in P^{32} treated patients.[89] Alkylating agents, such as busulfan or mechlorethamine, and other cytoxic agents, such as 6-mercaptopurine and demecolcine, have been used to inhibit the proliferation of marrow elements with some degree of success.

MYELOID METAPLASIA WITH MYELOFIBROSIS. Many descriptive terms have been applied to the group of illnesses characterized by a leukoerythroblastic anemia, enlargement of the spleen and liver as the result of myeloid metaplasia, and a patchy or generalized fibrosis of the bone marrow. The etiology of this syndrome remains obscure. There is an increased incidence of myeloid metaplasia among atom bomb survivors and others exposed to ionizing radiation.[89] One opinion relates this disorder to a leukemia of reticuloendothelial cells involving principally the marrow, spleen, and liver. At present, many group this condition with the myeloproliferative disorders largely because of their overlapping clinical features and because of the occasional transition from one form to another.[6, 90]

Clinical Presentation. Myeloid metaplasia, a rather uncommon illness, occurs in both sexes with equal frequency, generally after age 50. The primary complaints are weakness, easy fatigability, abdominal discomfort, and aching in the extremities, especially the legs. Less frequent symptoms are hemorrhage (skin and gastrointestinal tract), gout, and weight loss. Common physical abnormalities include pallor, ecchymoses or petechiae, striking splenomegaly, and more modest hepatomegaly. Icterus is present at times.

Although a few patients may have a normal or even increased red blood cell mass early in their illness, one of the most characteristic features of this disorder is the leukoerythroblastic anemia. The red cells show marked anisocytosis and poikilocytosis. Nucleated red cells are often seen. The reticulocyte count is normal or moderately elevated.

The white cell count is usually elevated, and the differential shows a shift to the left. All types of immature myelocytes may be encountered in the peripheral blood, including an occasional myeloblast. The alkaline phosphatase stain shows high or normal amounts of enzyme; rarely, low values are obtained. In most cases the platelet count is elevated and large, odd shapes are often encountered.

Attempts to secure marrow by aspiration may be unsuccessful. Biopsy techniques provide the means of demonstrating the myelofibrosis and hypocellularity present. Osteosclerosis may be seen also. These marrow changes may be patchy early in the disorder; later they may be generalized. Indeed, it is not uncommon to encounter foci of hematopoietic-cell hyperplasia. Biopsy or aspiration preparations from the spleen, the liver, and, rarely, the lymph nodes may show extensive extramedullary hematopoiesis (myeloid metaplasia).

X-rays of the bones in 25 to 50 percent of cases show a patchy irregular osteosclerosis. The cortex of the long bones is often thickened. Osteoporosis has also been noted. The blood uric acid level is elevated in about 50 percent of instances, at times to twice normal levels.

Diagnostic Considerations. The hallmarks of myeloid metaplasia with myeolofibrosis include a leukoerythroblastic anemia, marked splenomegaly and hepatomegaly with prominent myeloid metaplasia, and fibrosis or sclerosis of the bone marrow.

The diagnostic problems commonly confronted stem in part from the transition forms which exist between polycythemia vera, chronic myelocytic leukemia, and this disorder. In other instances, myeloid metaplasia must be distinguished from an aplastic or hypoplastic anemia. In addition, the physician must remain alert to the several possible causes of leukoerythroblastosis.

"Leukoerythroblastosis" is a term used to describe the presence of nucleated red cells and various immature myelocytic forms in the peripheral blood. When severe, this abnormality is striking, persistent, and usually accompanied by anemia. In its mildest forms there may be no anemia and only a few transiently circulating abnormal cells. Leu-

TABLE 2: Causes of Leukoerythroblastosis

I. Due to Abnormal Myeloproliferation Following
   A. Blood loss or hemolysis
   B. Nutritional deficiency anemias

II. Due to Myelophthisic Disorders
   A. Tuberculosis, especially disseminated
   B. Carcinomatosis (lung, breast, prostate)
   C. Xanthomatosis (Gaucher's disease and others)
   D. Lymphoma
   E. Myeloma
   F. Myeloproliferative disorders

III. Due to Severe Illness, Stress, Agonal States
   A. Infection
   B. Heart failure
   C. Uremia

koerythroblastosis may result from myeloproliferation or from myelophthisic disorders (Table 2). Carcinomatosis and other myelophthisic processes are the most frequently encountered causes of marked leukoerythroblastosis.

Clinical Course and Therapeutic Management. The average prognosis for life ranges from four to five years, but with good supportive care many patients live much longer. The main continuing clinical problems are those stemming from the anemia, the markedly enlarged spleen, or the hemorrhagic episodes.[6, 90, 93]

Symptoms resulting from severe anemia are relieved by transfusion. Androgens have improved the anemia in a few instances. Not infrequently, a hemolytic anemia develops, and the Coombs test may rarely become positive. Adrenal corticosteroids have benefited these individuals as well as a few others. Splenectomy has been performed to relieve this hemolytic problem. Thrombocytopenia with bleeding also develops on occasion and may be relieved by splenectomy. In these instances splenectomy has been performed de-

TABLE 3: Contrasting Clinical Features of the Chronic Myeloproliferative Disorders

CLINICAL FEATURES	POLYCYTHEMIA VERA	MYELOID METAPLASIA WITH MYELOFIBROSIS	CHRONIC MYELOCYTIC LEUKEMIA
Presentation	50% of cases	0	0
Pruritis	Plethora; erythema	0	Leukemic infiltrates
Skin lesions	When reduced hemoglobin exceeds 5 g per 100 ml	0	0
Ruddy cyanosis	Rare	Occasional	Rare
Jaundice	25–50% of cases	10–20% of cases	Occasional
Thromboses	Epistaxes, skin, central nervous system, gastrointestinal and joint bleeding	Especially gastrointestinal and skin bleeding	
Hemorrhage	50% of cases	Sometimes early	Occasional
Hypertension	Modest, 30–50% of cases	Most cases often large	Modest
Hepatomegaly	Usual, often large May be absent early	Often huge	Large
Splenomegaly	10–20% of cases	10–20% of cases	Occasional
Peptic ulcer	Approx. 5% of cases	5–10% of cases	Rare
Gout	Frequent	Approx. 20% of cases	Occasional
Bone pain	0	++	+++
Sternal tenderness			50–75% of cases
Course			
Average prognosis	10–20 years	4–5 years or more	3–4 years
Incidence of acute leukemia	1–10% of cases Related to P^{32} therapy	0	Terminal acute blastic crisis usual
Terminal events	Hemorrhage Thromboembolism Cardiovascular disorder Transition to myelofibrosis or chronic leukemia	Hemorrhage Thromboembolism Infection	Hemorrhage Infection

spite the fear attending removal of large amounts of hematopoietic tissue and the difficult operative and postoperative problems.[23] Following splenectomy, the liver enlarges progressively (myeloid metaplasia), and the general course of the disease continues.

External radiation has been directed at the spleen to relieve local symptomatology or to combat hemolytic anemia or thrombocytopenia. The potential danger of irradiating the areas of extramedullary hematopoiesis has been stressed also. Busulfan has induced a decrease in spleen size in some patients and an improvement in their hematologic status, but it may have adverse effects on the remaining hematopoietic tissue.

The immediate causes of death relate to underlying cardiovascular disease affected adversely by anemia, thromboembolic events, or hemorrhage. A transition to chronic myelocytic or acute myeloblastic leukemia may take place before death.

CONTRASTING CLINICAL FEATURES OF THE CHRONIC MYELOPROLIFERATIVE DISORDERS. Polycythemia vera, myeloid metaplasia with myelofibrosis, and chronic myelocytic leukemia are considered together primarily because they manifest a chronic uncontrolled proliferation of hematopoietic cells and because their clinical features often overlap or change in an interrelating fashion with the passage of time. Although certain basic morphologic resemblances are stressed, no implication with respect to etiology is in-

TABLE 4. Contrasting Hematologic Features of the Chronic Myeloproliferative Disorders

LABORATORY EXAMINATION	POLYCYTHEMIA VERA	MYELOID METAPLASIA WITH MYELOFIBROSIS	CHRONIC MYELOCYTIC LEUKEMIA
Hematocrit	Increased, often > 60	Decreased May show increase early	Decreased May be near normal early
Hemolysis	Normal erythrocyte survival	Occasional, with positive Coombs test	Normal erythrocyte survival
White Blood Cell Count	Usually 10–50,000; rarely > 100,000	Usually 20–50,000; rarely > 100,000	Generally elevated; often > 100,000
Differential	Normocytic red cells Occasional nucleated red cells Occasional myelocyte	Bizarre red cell shapes Frequent nucleated red cells Frequent myelocytes, occasional myeloblast	Normocytic red cells Occasional nucleated red cells Orderly shift to left with myelocytes, occasional myeloblast
Platelets	Usually elevated; > 400,000 in 50% of cases	Marked elevation, often bizarre forms May develop thrombocytopenic purpura	Usually elevated
Granulocyte Alkaline Phosphatase	Highest values	High to normal	Low to absent
Ph' Chromosome	0	0	+
Bone Marrow	Cellular Hyperplasia of all elements Read as normal in 50% of cases	Fibrosis and osteosclerosis Reticulum cell increase Patchy lesions early Focal areas of increased cellularity early	Myeloid hyperplasia At times increased red cells and megakaryocytes or patches of fibrosis
Myeloid Metaplasia	Rare foci in liver, spleen	Marked in spleen, liver At times in lymph nodes	0 (Myeloid infiltrates)
Blood (RBC) Volume	Increased	Normal (anemic)	Normal (anemic)
Blood Viscosity	Increased	Normal	Normal
Uric Acid	Increased	Increased in 50% of cases, at times twice normal	Increased
Immunoglobulins	Rare monoclonal gammopathy	Rare increase in γM + rheumatoid factor	—
X-rays of Bones	Usually normal	Patchy or diffuse osteosclerosis, thickened cortex, or osteoporosis in 25–50% of cases	Occasional subperiosteal new bone, osteolytic lesions, transverse lines at end of long bones

fibrosis as well as myeloid metaplasia in the spleen, although more frequently tuberculosis seems a secondary occurrence. However, present data do not permit definite conclusions concerning these or other suggested causal agents. The common proliferative abnormality seems neoplastic in character.[6, 89, 90]

The contrasting clinical features of these chronic myeloproliferative disorders are presented in Tables 3 and 4. Continuing clinical experience serves to emphasize the diversity of findings manifested by different patients and by the same patient during the course of his disease. A patient with classic polycythemia vera may after many months develop an increasing anemia and progressive enlargement of the spleen and liver. Appropriate biopsies may reveal myelofibrosis and extramedullary hematopoiesis associated with a leukoerythroblastic anemia. Similarly, an individual with polycythemia vera may develop all the features of chronic myelocytic eloblastic leukemia. This may evolve directly from the polycythemic status or may occur as a blast crisis in a person whose illness had previously become chronic myelocytic leukemia. The use of P^{32} therapy or other forms of radiation therapy has been linked to a tenfold increase in the incidence of acute leukemia.[89, 94]

Some patients with clear-cut myeloid metaplasia with myelofibrosis show transient polycythemia early in the course of their disease. The myeloid metaplasia syndrome seems unique among the myeloproliferative disorders because of the occasional associated occurrence of an autoimmune hemolytic anemia and the occasional associated development of thrombocytopenic purpura unrelated to therapy. A distinctive relationship appears to exist between the Ph' chromosome and chronic myelocytic leukemia, although exceptions have been reported.[6, 90]

59 The Lymphoreticular Proliferative Disorders

Lymphoid and reticuloendothelial tissue is located in many areas of the body. Apparent excessive production of lymphocytes, plasma cells, monocytes, and reticulum cells is a feature of several regional or systemic diseases. Although our current understanding of the homeostatic mechanisms regulating the basal rates of cell proliferation is limited, it seems clear that an increased production follows a variety of exogenous and endogenous stimuli and is related to the resulting immune responses. As with the myeloid disorders, the proliferative abnormalities of the lymphoreticular cells may be categorized further with respect to 1) their neoplastic or nonneoplastic characteristics, 2) the cell type(s) involved, 3) the duration of their course (acute or chronic), and 4) the associated immunologic response(s).[1, 6, 87, 88]

NONNEOPLASTIC LYMPHORETICULAR PROLIFERATION. Table 1 summarizes the variable immunoproliferative phenomena attending the majority of infectious diseases. In general, the mononuclear cell response occurs promptly after antigenic stimulation and subsides on removal of the stimulus. In acute bacterial (pyogenic) infections, lymphoreticular hyperplasia and plasmacytosis may follow the initial granulocyte response, whereas in most viral infections it is the primary event. The evidence of the associated immunologic responses (e.g., antibody titer, delayed cuta-

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STATEMENT OF [redacted] of P.O. 119, Innisfail, Queensland, residential address Menna Creek, (070) 65.3160

I, [redacted] state as follows:-

I went to Maralinga in 1957 when I was eighteen (18) (born 26.5.39). In 1957 I answered an advertisment in relation to a technical position with the Department of Supply. I was trained in Melbourne for approximately two (2) weeks from 25th July, 1957 to 9th August, 1957, in Health Physics procedures. I was actually stationed at Mount Clarence base via Woomera during the actual period of all the Antler tests at Maralinga, that is the tests on 14th September, 25th September and 9th October, 1957. The test sites were about 180 miles from Mount Clarence. My job entailed trying to position myself across the fallout path based on weather predictions and then when the fallout passed over, measuring and marking on maps what I had found. Many times the changes in wind meant that we completely missed the fallout. Other times however, it resulted in our being directly under where the heaviest fallout fell. In one case I ended up with some much fallout in my hair that it was impossible to wash it so my hair had to be all cut off. One of the things that disturbed me was the fact that quite often I would find small patches of much higher

radiation than background in areas where none of the fallout from the tests we were working had gone. It was not until much later that I was able to find out that in fact it was the path where the fallout from 1956 series of test had gone. Another part of my job at Mount Clarence was the collection of sheep thyroid glands for the measure of the Iodine 131. We would collect the thyroid glands from the sheep at a time after the fall-out was supposed to have fallen. These were then sent to Maralinga by plane and were measured to see just how much Iodine 131 the sheep had eaten. At the time there was a severe drought in the area and consequently no grass so much so that many of the graziers were paying to have sheep shipped off their properties. As a consequence some of these sheep had not eaten for weeks and of course little or no Iodine 131 was found.

About one week after the antler trials had concluded I was transferred to Maralinga to the Health Physics team under a [redacted] My work involved the control of personnel entering the test area, decontamination of personnel and clothing and radiation surveys of the actual bomb sites. I ran the laundry where radio-active clothing was washed for some time at Maralinga.

The measurements I carried out at the bomb sites involved danger because there was still high

radiation levels at the site and it was important to do these as quickly as possible to reduce the exposure time.

However, we were working a lot of overtime at the time and it was not uncommon to almost triple ones normal pay. The overtime was only available when working in the forward zone however and once our film badges and other personal monitoring devices showed we had reached a certain level we were restricted to the rear-ward zone with a consequent loss of overtime. Therefore, it was common practice among the people involved including myself that as soon as you knew you had reached the allowed level you had to leave the film badges and the dosimeters in the Land Rover so they did not register. At the time this seemed to be just a big game and the overtime was terrific. Because of this practice the recorded levels of radiation measured were only a fraction of what I actually received. I recall that on one occasion my film badge was so black that the densitometer being used could not even read how much radiation I had received. I received my major radiation exposure however during the surveys of a bomb site about six (6) months after the actual explosion. To allow repeatibility in the measurements of the craters, a set of twelve lines radiated out from the actual ground zero point. Wooden marker pegs were placed at 100 foot intervals along

these lines, and the measurements were taken at these pegs. This allowed us to get a reasonably good idea of what was going on, but it suffered from the fact that the area between the radiating lines never got measured at all. I had been surveying this crater for some months, and one day when I was out by myself doing a survey of this crater, I noticed a few anomalous readings. I decided to do a bit more investigation, and found that there was a very much higher level of radiation in an area between the normally surveyed lines. The level of radiation was such that the instrument I was using could not measure it. However there was a mode in which these instruments had been found to work, that although it could not give calibrated readings, it could show relative differences. Using the instrument in this mode, I found that this high level of radiation was coming from a number of quite discrete sources, and by using my right foot to move the dirt about I was finally able to discover that the radiation was coming from some very small metallic looking particles, so by using my foot again I was able to locate about 20-30 of these and put them into one of the tobacco tins which we used for sample collection. I put this tin in the back of the Land Rover just behind me and drove back the thirty (30) odd miles to the Health Physics Labs. When I pulled up, the rest

of the staff were already outside waiting even though I had not notified them of my arrival. The reason for them being there was that as I approached, all the instruments in the lab went haywire. We were able to quickly ascertain that these pellets were much more powerful than anything we had ever previously detected. The only way we were able to measure the pellets was by leaving them outside the building, and measuring them from outside. We were soon able to establish by use of a Pearson scanning spectrometer that the pellets were Cobalt 60 with a half-life of 5.2 years. Cobalt 60 was one of the last things we expected in the normal fallout, and certainly not at this order of level. Quickly we got in touch with the [redacted] at [redacted] and immediately a heavy security blanket went into effect. I was interviewed by a security officer and it was impressed upon me not to speak to any-one about this at all, particularly any Australians, no matter what their position at Maralinga. A major effort was immediately started to try to collect as much of this material as possible, but there were problems because the level of radiation was so high that it was obvious that the existing Health Physics Group Personnel could not do it because by this time we were already over our limits. At this time there were a number of Australian Services personnel

who were supposed to be there on a training course in radition detection. These people could not be told what they were working with because of the security blanket, however I made up some long scoops by tying jam tins onto wooden handles, and I showed them how to use these to collect the particles. By this time, special aircraft had been flown out from the U.K. with large lead containers, and the particles were placed into these and then flown immediately back to the U.K. As the person who actually discovered the particles, and was involved in their recovery, I was under the distinct impression that the British authorities did not want the Australian Government to know anything at all about what had happened. This Cobalt accident happened in about 1958 to the best of my recollection and the Cobalt was related to the Antler series.

At the time this actually occurred I did not seem to have suffered from this radiation exposure. However at the end of June 1959 I left Maralinga to take up a position with the [redacted] at [redacted] in the U.K., and some months later took up another position in London. At this time I started to suffer various fever symptoms and went to different hospitals in London for tests. The doctors in each case said the symptoms were similar to those of malaria, but blood tests showed that I

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could try to determine if my marrow was still detiorating or was steady. The final conclusion reached was that marrow damage had occurred, but did not seem to be getting worse. It was however conclusively proved that as a result of the marrow damage I had a permanently low blood count.

Also about this time, I started to suffer a lot from lack-of-breath. Even just walking up a flight of steps would leave me winded, even though tests done at James Cook University in Townsville showed that I had a larger than normal lung capacity. A Townsville doctor who examined me a number of times, and who obtained my whole medical history from previous doctors, finally concluded that it was probably a form of emphysema caused by the radiation.

By 1981 the radiation had not affected me to the extent where it had incapacitated me. However the side effects of it had almost resulted in my death from other causes. For example, in early 1977 I went to Bali for holidays and contacted salmonella-para typhoid. I was flown back to Australia unconcious and put into quarantine hospital. Because of the advanced state of the disease I was treated with a rather dangerous drug Chloreamphenicol. Normally this drug is only used until such time as the blood count starts to rise. (A lowered blood-count is one of the symptoms of Salmonella-para typhoid).

However, due to my now permanently low blood count, and the fact that I was unconscious and unable to tell the doctors that, I was treated with the drug for a much longer period than normal. When I finally became conscious and was able to tell the doctors about my radiation damage they ceased using it immediately, however the damage had already been done. Seven (7) years later I still suffer gastric complaints from the overdose of Chloramphenicol. This problem of complicating medical diagnosis is one of the main problems that I have had to live with in the past. Since 1981 my health has further deteriorated and I am no longer able to work full-time.

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The DC-12 Building was part of the DCRB Group of Buildings, it was associated with the Minor Trials Kittens Tests. DC-12 Building was basically a very large hot-box, a very large and very thick lead lined box with long manipulator arms that we used from outside. It had glass windows, possibly a couple of feet thick, it was a bit difficult to tell but it was very heavily shielded. This building was used by the teams who came from the U.K. for the preparation of the Minor Trial during which time, while they were out, Australians were not allowed near the building. During the period between Minor Trials it was our responsibility to maintain this building and particularly the filtering system associated with it because of the nature of the materials that they were using inside it, most probably Plutonium. There was a lot of Radon Gas given off, this was withdrawn from the hot-box and out through some filters and a large metal ducting system. The idea of this was that the Radon had a quite short half-life and by keeping the air as it was sucked through in this long series of chambers it spent enough time in there for a lot of the Radon to decay back into a solid material, one of the decay products of possibly lead or some other isotope. There were two (2) large filters which we used to filter out a lot of the solid materials, at one stage it became necessary to replace these

two (2) filters because they had gathered enough material that air flow through them was being hindered. This was measured because there was a water pressure gauge type of thing across it which essentially measured the pressure drop across it and as the filter became more and more filled with material so the pressure drop across it increased and it got to the point where the filter had to be replaced. These filters were large steel boxes with flanges at either end where they connected on to the rest of the duct work. It was necessary to use a crane to come up because they were so heavy but it was also necessary to unbolt them from the rest of the duct work. This meant that one period after unbolting and as they were being lifted out that in fact the ends of the filters were exposed to the atmosphere. People had to be there to unbolt them and that at one stage was my job. I worked with an English Chemist whose name I cannot recall at the moment, his name was [redacted] We had no proper breathing equipment available and so I had to jury-rig some equipment. For the first filter change episode I obtained a couple of small air compressors that were used in the yellow garage and these were coupled through long lengths of pressure hose to gas masks and [redacted] and I donned a gas mask each and began one of us working at either end, to unbolt the filters. During tests

beforehand it appeared that there was sufficient air being supplied by the compressors, however, after the filters had actually been unbolted and in the process of trying to manipulate them out of the major duct line, the physical effort involved meant that our breathing rate was increased quite dramatically and it became quite apparent that there was not sufficient air being delivered by the compressors and at one stage I started to breathe in and at that point my head was right level and only inches away from the input side of the filter and I started to get leaks in the gas mask because of the insufficient air and I started to breathe in the air that was in the vicinity of the mouth of the filter. Eventually we were able to get it out and the new filter bolted in. For the second filter, knowing the problems we had had with the small air compressors which were basically designed for low volume but high pressure, I decided to use something that was high volume low pressure and finally settled on vacuum cleaners so we had two (2) vacuum cleaners which were coupled on there outlet side by long large diameter plastic tubing ~~tubes~~ plate about an inch and a half in diameter again to the gas masks. Preliminary testing seemed to indicate that this was much more satisfactory, that there would be an adequate supply of air, however, again when we are at that critical stage when the filter

had actually been unbolted and was half way out, I found a problem with this plastic tubing we were using, which actually came in the form of a flat strip on a roll. The plastic pipe kinked affectively cutting off my air supply completely. It became necessary for me at one stage to completely remove the gas mask in order to breathe. I attempted to point my head away from the entrance of the filter in order to reduce any radio-active materials that I might enhail. We had to operate quickly and seal the ends of the filter being removed and then wrap it up and they were later disposed of. The point of this is that it demonstrates the lack of correct equipment that was available. We were required to perform certain operations for which there was no correct equipment available and in many cases jury-rigged equipment turned out to be safety hazards.

I also recall the problems that we experienced with the disposal of radio-active wastes in the RB-DC area. Contaminated vehicles from the front line and other materials brought back for experiment became a problem. At one stage it became necessary to do something active about the accumulated waste that we had on hand. I was given the job of disposing of this and starting a graveyard. [redacted] in conjunction with Range Authorities determined an area that was to be used and Army staff dug a

large pit there for which we would be able to use to put the drums in and they would be progressively covered over as we filled up the area. I was given the job of disposing of quite a number of these materials, some of them being wastes from the DC-12 Building, where the Minor Trials Preparations went on and others were glazing and other such materials from the fordward area. The technique I adopted was to parcel these materials up into a plastic bag and use the heat sealer from DC-12 to seal the plastic bags. I would then put this plastic bag inside a large milo tin of which we had plenty. I would then take a 44 gallon drum and cut out the top of it. I would then mix up concrete and fill the 44 gallon drum to about half way and then wait for that to set, sometimes a period of 3-4 days. I would then place the milo tin in the middle of the 44 gallon drum on top of the hardened concrete and then mix up another batch of concrete and pour it over the top until the 44 gallon drum was full. At some stage later the Army people would come in with the yellow crane and take it to the graveyard where it would be deposited. My worries since have revolved around the fact that these 44 gallon drums may well have corroded. The juncture between the two layers of concrete, the top layer was poured last and the bottom layer which was poured some time several days beforehand,

this gap may well have allowed moisture from the ground to penetrate through to the milo tins, if the milo tins in turn rusted or in other ways corroded it may well be possible for some of these radio-active wastes to have been leached out into the soil. In retrospect it was a most unsatisfactory method of waste disposal that in the absence of any better knowledge or better techniques and given that it was up to my own what-have-you to come up with some method, that was the method I adopted.

DATED this day of , 1984.

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[redacted]

BADGES.

When I joined the group at Maralinga post-Antler, we were requesthed the job of maintaining film badge records of radiation. The film badges issued were some ancient old stock much of it already past its expiry date. These were issued at the front line caravans to personnel going into the blast areas. Many of them were collected for processing, but many of them were 'lost' or souvineered. The badges were taken back to the RB labs where we developed them using some old x-ray developer. I had never developed film in my life before but after one showing was expected to be able to do it. The dark room used for developing had no fine temperature control and usually ran very cold. The method of controlling the temperature of the developing solutions was to place the beaker containing the developer into a larger container and put a mixture of hot and cold water into the outer container until the correct temperature was obtained. After developing the film badges were then read on an optical densitometer. This instrument just measured the optical density of the film in some arbitary units. These were then converted to a radiation measurement using a curve on a piece of graph paper stuck on the side of the machine. There was NO calibration for the densitomer itself. Whenever we had to change a bulb in it the procedure was to adjust a potentiometer on it until it gave a certain reading. Where the information on what this reading really was I was never able to find out. neither was I ever able to find out where the conversion graph came from. Suffice it to say that we used this system despite many changes in production batches of fim badges, changes in the chemicals used for developing, etc. without any recalibration of the equipment. The curve on the graph paper only covered a fairly narrow range of the densities measureable by the densitomer. In some cases the film badges were so dark that the optical density reading obtained was completely off the graph. The graph was already non-linear but despite this, when this problem occurred we would just guess a value and record this. This state of affairs continued until after I discovered the Cobalt 60 at Taddje. Using various other sources available we were able to estimate the strength of one of the Cobalt pellets. This was then placed in a jury-rigged arrangement of laboratory stands in one of the compounds with an array of film badges at various distances from the source. By this method we were able to relate radiation received to the optical densitomer readings and thus produce a new calibration curve.

THYROID IODINE 131 MEASUREMENTS

While I was with the L.R.D.U. group at Mt. Clarence, part of our function was to collect thyroid glands from sheep in what was thought to be the general fallout path. Several of the L.R.D.U. members had in fact had an additional 1 weeks training in Melbourne following the 2 week general course which we all undertook. At tat time in central Australia there had in fact been a drought for about 2 years in places. In some instances sheep farmers were paying to have dying sheep removed from their property so that they did not foul the few remaining sources of water. Imagine their delight when we came along and offered them $10 (5 pounds) for the thyroid glands of just on sheep. In many cases the farmer would choose the sheep closest to death anyway. The point of the matter is that the project was based on the assumption that the sheep would have been grazing on grass and thus would have ingested any Iodine 131 which may have fallen. If there is no grass to eat then no matter how much Iodine 131 may have fallen there will be no sign of it in the thyroid glands. My belief is that the project may have been dreamed up by U.K. scientists who had absolutely no idea of the Australian outback. I also believe that the only accurate readings of Iodine 131 that may have been obtained was when the fallout from one of the earlier tests went south-east towards Adelaide instead of north-east and thus fell on reasonable pastures where sheep were in fact grazing. These measurements unfortunately were not done by Maralinga scientists and were thus discounted.

FIBRE DOSIMETERS.

Quartz fibre dosimeters were used in the forward area with the of being able to get instantaneous readings of radiation received. 2 different types of instruments were in use, one with a 10 times higher range than the other. If my memory serves me correct I believe that one may have been 0-50 and the other 0-500. In the the post-test phase when radiation levels had reduced somewhat only the 0-50 instruments were supposed to be issued but because of the limited number of them available quite often 0-500 instruments were issued which would not have registered anything. The quartz fibre dosimeters in use had a fragile quartz fibre assembly and microscope optical assembly to read it with. Because of the fragile nature of them many of them were in a damaged condition e.g. either the fibre could not be seen at all, or there might be no movement of the fibre between the charged and discharged states. In many cases I found that some of the personnel working in the forward area would demand a dosimeter even though no workable ones were available because many of them were under the mistaken impression that the wearing of them prevented on from receiving any radiation. The dosimeters were electrically charged by means of a battery operated charging unit. The majority of the charging units were defunct because of different electronic faults, and it was difficult to obtain good batteries for the remainder. A large chest freezer unit was kept at the RB unit and this contained hundreds of batteries of different types. Most of the batteries were past their expiry date and had suffered water damage. The general procedure was to grab a handful of the batteries for the dosimeter chargers and hope that amongst them one could find one that still had enough charge to operate. In many cases the radiation levels recorded for persons working in the forward was 'guesstimated' based on the known levels in the general area where they were supposed to be working, and perhaps correlated with the readings of someone who might have been lucky to actually have a working dosimeter.

HOT SPOTS.

While at Mt. Clarence with the L.R.D.U. during the actual Antler trials I was in charge of a small group whose duty was to drive to the expected path of the fallout and take measurements as the fallout passed over. In some cases the wind changed and we were nowhere near where the fallout actually fell. In other cases we were directly underneath. In fact in one case I received fallout in my hair which even after about 3 washings could not be removed so the camp barber just gave me a crew cut which solved the problem. In a couple of instances while we actually in place and waiting for the fallout to arrive I decided on my own initiative to do some general background readings using a G.M. counter. Imagine my suprise when I found occasional spots where the count was already about 100 times greater that the general background. After some investigation I established that this was usually associated with clumps of spinifex, and not on the general cleared areas. My assumption was that it must have been the results of fallout from an earlier series of tests, perhaps the Buffalo series, and that wind action might have resulted in the concentrating of the actual fallout material in the vicinity of the spinifex plants. An alternative possibility occurred to me much later. It was noticed that about a year after the tests by which time there had been some rain, that when viewed from the air there was a definite increase in the vegetation in the areas where fallout had actually occurred. This was explained to me by one person who pointed out that the whole area was known to be deficient in rare earths often necessary for plant growth and that perhaps the fallout was in fact providing the necessary rare earths. Suprisingly enough this fact was bought to my attention again only about 6 years ago when I was working with a scientist whose Ph.D. thesis in botany was based in the central Australian region. In his thesis he drew attention to certain clumping of vegetation which he had observed in the area. He was not aware that atomic fallout had been over those areas and was quite suprised when I bought it to his attention. He agreed that this was the only logical explanation which satisfied his observations. This would tend to suggest then that even 20 years after the event the affect of atomic fallout in central Australia can still be observed.

DOCUMENT

GROUP

SEPARATOR SHEET

[redacted] of P.O. 119, Innisfail, Queensland, residential address Menna Creek, (070) 65.3160

I, [redacted] state as follows:-

I went to Maralinga in 1957 when I was eighteen (18) (born 26.5.39). In 1957 I answered an advertisment in relation to a technical position with the Department of Supply. I was trained in Melbourne for approximately two (2) weeks from 25th July, 1957 to 9th August, 1957, in Health Physics procedures. I was actually stationed at Mount Clarence base via Woomera during the actual period of all the Antler tests at Maralinga, that is the tests on 14th September, 25th September and 9th October, 1957. The test sites were about 180 miles from Mount Clarence. My job entailed trying to position myself across the fallout path based on weather predictions and then when the fallout passed over, measuring and marking on maps what I had found. Many times the changes in wind meant that we completely missed the fallout. Other times however, it resulted in our being directly under where the heaviest fallout fell. In one case I ended up with some much fallout in my hair that it was impossible to wash it so my hair had to be all cut off. One of the things that disturbed me was the fact that quite often I would find small patches of much higher

radiation than background in areas where none of the fallout from the tests we were working had gone. It was not until much later that I was able to find out that in fact it was the path where the fallout from 1956 series of test had gone. Another part of my job at Mount Clarence was the collection of sheep thyroid glands for the measure of the Iodine 131. We would collect the thyroid glands from the sheep at a time after the fall-out was supposed to have fallen. These were then sent to Maralinga by plane and were measured to see just how much Iodine 131 the sheep had eaten. At the time there was a severe drought in the area and consequently no grass so much so that many of the graziers were paying to have sheep shipped off their properties. As a consequence some of these sheep had not eaten for weeks and of course little or no Iodine 131 was found.

About one week after the antler trials had concluded I was transferred to Maralinga to the Health Physics team under a [redacted] My work involved the control of personnel entering the test area, decontamination of personnel and clothing and radiation surveys of the actual bomb sites. I ran the laundry where radio-active clothing was washed for some time at Maralinga.

The measurements I carried out at the bomb sites involved danger because there was still high

radiation levels at the site and it was important to do these as quickly as possible to reduce the exposure time.

However, we were working a lot of overtime at the time and it was not uncommon to almost triple ones normal pay. The overtime was only available when working in the forward zone however and once our film badges and other personal monitoring devices showed we had reached a certain level we were restricted to the rear-ward zone with a consequent loss of overtime. Therefore, it was common practice among the people involved including myself that as soon as you knew you had reached the allowed level you had to leave the film badges and the dosimeters in the Land Rover so they did not register. At the time this seemed to be just a big game and the overtime was terrific. Because of this practice the recorded levels of radiation measured were only a fraction of what I actually received. I recall that on one occasion my film badge was so black that the densitometer being used could not even read how much radiation I had received. I received my major radiation exposure however during the surveys of a bomb site about six (6) months after the actual explosion. To allow repeatibility in the measurements of the craters, a set of twelve lines radiated out from the actual ground zero point. Wooden marker pegs were placed at 100 foot intervals along

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DATED this day of , 1984.

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[redacted]

'AIDGES.

When I joined the group at Maralinga post-Antler, we were ?athed the job of maintaining film badge records of radiation. The badges issued were some ancient old stock much of it already past expiry date. These were issued at the front line caravans to ?onnel going into the blast areas. Many of them were collected for ?cessing, but many of them were 'lost' or souvineered. The badges were ?en back to the RB labs where we developed them using some old x-ray ?eloper. I had never developed film in my life before but after one ?owing was expected to be able to do it. The dark room used for ?veloping had no fine temperature control and usually ran very cold. ?he method of controlling the temperature of the developing solutions was to place the beaker containing the developer into a larger container and put a mixture of hot and cold water into the outer container until the correct temperature was obtained. After developing the film badges were then read on an optical densitometer. This instrument just measured the optical density of the film in some arbitary units. These were then converted to a radiation measurement using a curve on a piece of graph paper stuck on the side of the machine. There was NO calibration for the densitomer itself. Whenever we had to change a bulb in it the procedure was to adjust a potentiometer on it until it gave a certain reading. Where the information on what this reading really was I was never able to find out. Neither was I ever able to find out where the conversion graph came from. Suffice it to say that we used this system despite many changes in production batches of fim badges, changes in the chemicals used for developing, etc. without any recalibration of the equipment. The curve on the graph paper only covered a fairly narrow range of the densities measureable by the densitomer. In some cases the film badges were so dark that the optical density reading obtained was completely off the graph. The graph was already non-linear but despite this, when this problem occurred we would just guess a value and record this. This state of affairs continued until after I discovered the Cobalt 60 at Tadje. Using various other sources available we were able to estimate the strength of one of the Cobalt pellets. This was then placed in a jury-rigged arrangement of laboratory stands in one of the compounds with an array of film badges at various distances from the source. By this method we were able to relate radiation received to the optical densitomer readings and thus produce a new calibration curve.

THYROID IODINE 131 MEASUREMENTS

While I was with the L.R.D.U. group at Mt. Clarence, part of our function was to collect thyroid glands from sheep in what was thought to be the general fallout path. Several of the L.R.D.U. members had in fact had an additional 1 weeks training in Melbourne following the 2 week general course which we all undertook. At tet time in central Australia there had in fact been a drought for about 2 years in places. In some instances sheep farmers were paying to have dying sheep removed from their property so that they did not foul the few remaining sources of water. Imagine their delight when we came along and offered them $10 (5 pounds) for the thyroid glands of just one sheep. In many cases the farmer would choose the sheep closest to death anyway. The point of the matter is that the project was based on the assumption that the sheep would have been grazing on grass and thus would have ingested any Iodine 131 which may have fallen. If there is no grass to eat then no matter how much Iodine 131 may have fallen there will be no sign of it in the thyroid glands. My belief is that the project may have been dreamed up by U.K. scientists who had absolutely no idea of the Australian outback. I also believe that the only accurate readings of Iodine 131 that may have been obtained was when the fallout from one of the earlier tests went south-east towards Adelaide instead of north-east and thus fell on reasonable pastures where sheep were in fact grazing. These measurements unfortunately were not done by Maralinga scientists and were thus discounted.

FILM DOSIMETERS.

Quartz fibre dosimeters were used in the forward area with the of being able to get instantaneous readings of radiation received. o different types of instruments were in use, one with a 10 times igher range than the other. If my memory serves me correct I believe that one may have been 0-50 and the other 0-500. In the the post-test phase when radiation levels had reduced somewhat only the 0-50 instruments were supposed to be issued but because of the limited number of them available quite often 0-500 instruments were issued which would not have registered anything. The quartz fibre dosimeters in use had a fragile quartz fibre assembly and microscope optical assembly to read it with. Because of the fragile nature of them many of them were in a damaged condition e.g. either the fibre could not be seen at all, or there might be no movement of the fibre between the charged and discharged states. In many cases I found that some of the personnel working in the forward area would demand a dosimeter even though no workable ones were available because many of them were under the mistaken impression that the wearing of them prevented on from receiving any radiation. The dosimeters were electrically charged by means of a battery operated charging unit. The majority of the charging units were defunct because of different electronic faults, and it was difficult to obtain good batteries for the remainder. A large chest freezer unit was kept at the RB unit and this contained hundreds of batteries of different types. Most of the batteries were past their expiry date and had suffered water damage. The general procedure was to grab a handful of the batteries for the dosimeter chargers and hope that amongst them one could find one that still had enough charge to operate. In many cases the radiation levels recorded for persons working in the forward was 'guesstimated' based on the known levels in the general area where they were supposed to be working, and perhaps correlated with the readings of someone who might have been lucky to actually have a working dosimeter.

HOT SPOTS.

While at Mt. Clarence with the L.R.D.U. during the actual Antler trials I was in charge of a small group whose duty was to drive to the expected path of the fallout and take measurements as the fallout passed over. In some cases the wind changed and we were nowhere near where the fallout actually fell. In other cases we were directly underneath. In fact in one case I received fallout in my hair which even after about 3 washings could not be removed so the camp barber just gave me a crew cut which solved the problem. In a couple of instances while we actually in place and waiting for the fallout to arrive I decided on my own initiative to do some general background readings using a G.M. counter. Imagine my suprise when I found occasional spots where the count was already about 100 times greater that the general background. After some investigation I established that this was usually associated with clumps of spinifex, and not on the general cleared areas. My assumption was that it must have been the results of fallout from an earlier series of tests, perhaps the Buffalo series, and that wind action might have resulted in the concentrating of the actual fallout material in the vicinity of the spinifex plants. An alternative possibility occurred to me much later. It was noticed that about a year after the tests by which time there had been some rain, that when viewed from the air there was a definite increase in the vegetation in the areas where fallout had actually occurred. This was explained to me by one person who pointed out that the whole area was known to be deficient in rare earths often necessary for plant growth and that perhaps the fallout was in fact providing the necessary rare earths. Suprisingly enough this fact was bought to my attention again only about 6 years ago when I was working with a scientist whose Ph.D. thesis in botany was based in the central Australian region. In his thesis he drew attention to certain clumping of vegetation which he had observed in the area. He was not aware that atomic fallout had been over those areas and was quite suprised when I bought it to his attention. He agreed that this was the only logical explanation which satisfied his observations. This would tend to suggest then that even 20 years after the event the affect of atomic fallout in central Australia can still be observed.

DOCUMENT

GROUP

SEPARATOR SHEET

STATEMENT OF [redacted] of [redacted]

P.O. 119, Innisfail, Queensland, residential address Menna Creek, (070) 65.3160

I, [redacted] state as follows:-

I went to Maralinga in 1957 when I was eighteen (18) (born 26.5.39). In 1957 I answered an advertisment in relation to a technical position with the Department of Supply. I was trained in Melbourne for approximately two (2) weeks from 25th July, 1957 to 9th August, 1957, in Health Physics procedures. I was actually stationed at Mount Clarence base via Woomera during the actual period of all the Antler tests at Maralinga, that is the tests on 14th September, 25th September and 9th October, 1957. The test sites were about 180 miles from Mount Clarence. My job entailed trying to position myself across the fallout path based on weather predictions and then when the fallout passed over, measuring and marking on maps what I had found. Many times the changes in wind meant that we completely missed the fallout. Other times however, it resulted in our being directly under where the heaviest fallout fell. In one case I ended up with some much fallout in my hair that it was impossible to wash it so my hair had to be all cut off. One of the things that disturbed me was the fact that quite often I would find small patches of much higher

radiation than background in areas where none of the fallout from the tests we were working had gone. It was not until much later that I was able to find out that in fact it was the path where the fallout from 1956 series of test had gone. Another part of my job at Mount Clarence was the collection of sheep thyroid glands for the measure of the Iodine 131. We would collect the thyroid glands from the sheep at a time after the fall-out was supposed to have fallen. These were then sent to Maralinga by plane and were measured to see just how much Iodine 131 the sheep had eaten. At the time there was a severe drought in the area and consequently no grass so much so that many of the graziers were paying to have sheep shipped off their properties. As a consequence some of these sheep had not eaten for weeks and of course little or no Iodine 131 was found.

About one week after the antler trials had concluded I was transferred to Maralinga to the Health Physics team under a [redacted] My work involved the control of personnel entering the test area, decontamination of personnel and clothing and radiation surveys of the actual bomb sites. I ran the laundry where radio-active clothing was washed for some time at Maralinga.

The measurements I carried out at the bomb sites involved danger because there was still high

radiation levels at the site and it was important to do these as quickly as possible to reduce the exposure time.

However, we were working a lot of overtime at the time and it was not uncommon to almost triple ones normal pay. The overtime was only available when working in the forward zone however and once our film badges and other personal monitoring devices showed we had reached a certain level we were restricted to the rear-ward zone with a consequent loss of overtime. Therefore, it was common practice among the people involved including myself that as soon as you knew you had reached the allowed level you had to leave the film badges and the dosimeters in the Land Rover so they did not register. At the time this seemed to be just a big game and the overtime was terrific. Because of this practice the recorded levels of radiation measured were only a fraction of what I actually received. I recall that on one occasion my film badge was so black that the densitometer being used could not even read how much radiation I had received. I received my major radiation exposure however during the surveys of a bomb site about six (6) months after the actual explosion. To allow repeatibility in the measurements of the craters, a set of twelve lines radiated out from the actual ground zero point. Wooden marker pegs were placed at 100 foot intervals along

these lines, and the measurements were taken at these pegs. This allowed us to get a reasonably good idea of what was going on, but it suffered from the fact that the area between the radiating lines never got measured at all. I had been surveying this crater for some months, and one day when I was out by myself doing a survey of this crater, I noticed a few anomalous readings. I decided to do a bit more investigation, and found that there was a very much higher level of radiation in an area between the normally surveyed lines. The level of radiation was such that the instrument I was using could not measure it. However there was a mode in which these instruments had been found to work, that although it could not give calibrated readings, it could show relative differences. Using the instrument in this mode, I found that this high level of radiation was coming from a number of quite discrete sources, and by using my right foot to move the dirt about I was finally able to discover that the radiation was coming from some very small metallic looking particles, so by using my foot again I was able to locate about 20-30 of these and put them into one of the tobacco tins which we used for sample collection. I put this tin in the back of the Land Rover just behind me and drove back the thirty (30) odd miles to the Health Physics Labs. When I pulled up, the rest

of the staff were already outside waiting even though I had not notified them of my arrival. The reason for them being there was that as I approached, all the instruments in the lab went haywire. We were able to quickly ascertain that these pellets were much more powerful than anything we had ever previously detected. The only way we were able to measure the pellets was by leaving them outside the building, and measuring them from outside. We were soon able to establish by use of a Pearson scanning spectrometer that the pellets were Cobalt 60 with a half-life of 5.2 years. Cobalt 60 was one of the last things we expected in the normal fallout, and certainly not at this order of level. Quickly we got in touch with the [redacted] at [redacted] and immediately a heavy security blanket went into effect. I was interviewed by a security officer and it was impressed upon me not to speak to any-one about this at all, particularly any Australians, no matter what their position at Maralinga. A major effort was immediately started to try to collect as much of this material as possible, but there were problems because the level of radiation was so high that it was obvious that the existing Health Physics Group Personnel could not do it because by this time we were already over our limits. At this time there were a number of Australian Services personnel

who were supposed to be there on a training course in radition detection. These people could not be told what they were working with because of the security blanket, however I made up some long scoops by tying jam tins onto wooden handles, and I showed them how to use these to collect the particles. By this time, special aircraft had been flown out from the U.K. with large lead containers, and the particles were placed into these and then flown immediately back to the U.K. As the person who actually discovered the particles, and was involved in their recovery, I was under the distinct impression that the British authorities did not want the Australian Government to know anything at all about what had happened. This Cobalt accident happened in about 1958 to the best of my recollection and the Cobalt was related to the Antler series.

At the time this actually occurred I did not seem to have suffered from this radiation exposure. However at the end of June 1959 I left Maralinga to take up a position with the [redacted] at [redacted] in the U.K., and some months later took up another position in London. At this time I started to suffer various fever symptoms and went to different hospitals in London for tests. The doctors in each case said the symptoms were similar to those of malaria, but blood tests showed that I

was not suffering from malaria. I returned to Australia in the early 1960's and worked first in Sydney, then at Woomera, that at Brisbane. During this time I started to suffer from extreme pain in my right foot, so severe that it would keep me awake at nights, and sometimes I could not sleep for 2 or 3 nights at a time. I went to many different doctors about the problem in my foot, and many x-rays were taken but nothing wrong physically could be found. Various suggesions were made about possible gout, but again blood tests proved that it was not gout. The final pointer to the possible problem came when I was in a Brisbane Hospital for another reason completely. The hospital had a scheme of blood tests for all patients, possibly as part of a V.D. detection campaign. My tests showed up abnormal so many further tests were done. Again symptoms similar to malaria must have been found because the doctor who was doing all the tests on me actually asked, in order to rule out all possibility if perhaps I'd ever had malaria. Finally he just happened to ask if I had ever been exposed to radiation, and when I said "yes", he immediately said that could most probably be the cause. He then arranged for marrow samples to be taken from the bones in my hips, and these showed marrow damage. I had to return to the hospital every couple of months from then on so that they

could try to determine if my marrow was still detiorating or was steady. The final conclusion reached was that marrow damage had occurred, but did not seem to be getting worse. It was however conclusively proved that as a result of the marrow damage I had a permanently low blood count.

Also about this time, I started to suffer a lot from lack-of-breath. Even just walking up a flight of steps would leave me winded, even though tests done at James Cook University in Townsville showed that I had a larger than normal lung capacity. A Townsville doctor who examined me a number of times, and who obtained my whole medical history from previous doctors, finally concluded that it was probably a form of emphysema caused by the radiation.

By 1981 the radiation had not affected me to the extent where it had incapacitated me. However the side effects of it had almost resulted in my death from other causes. For example, in early 1977 I went to Bali for holidays and contacted salmonella-para typhoid. I was flown back to Australia unconcious and put into quarantine hospital. Because of the advanced state of the disease I was treated with a rather dangerous drug Chloreamphenicol. Normally this drug is only used until such time as the blood count starts to rise. (A lowered blood-count is one of the symptoms of Salmonella-para typhoid).

However, due to my now permanently low blood count, and the fact that I was unconscious and unable to tell the doctors that, I was treated with the drug for a much longer period than normal. When I finally became conscious and was able to tell the doctors about my radiation damage they ceased using it immediately, however the damage had already been done. Seven (7) years later I still suffer gastric complaints from the overdose of Chloramphenicol. This problem of complicating medical diagnosis is one of the main problems that I have had to live with in the past. Since 1981 my health has further deteriorated and I am no longer able to work full-time.

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[redacted]

BADGES.

When I joined the group at Maralinga post-Antler, we were requesthed the job of maintaining film badge records of radiation. The film badges issued were some ancient old stock much of it already past its expiry date. These were issued at the front line caravans to personnel going into the blast areas. Many of them were collected for processing, but many of them were 'lost' or souvineered. The badges were taken back to the RB labs where we developed them using some old x-ray developer. I had never developed film in my life before but after one showing was expected to be able to do it. The dark room used for developing had no fine temperature control and usually ran very cold. The method of controlling the temperature of the developing solutions was to place the beaker containing the developer into a larger container and put a mixture of hot and cold water into the outer container until the correct temperature was obtained. After developing the film badges were then read on an optical densitometer. This instrument just measured the optical density of the film in some arbitary units. These were then converted to a radiation measurement using a curve on a piece of graph paper stuck on the side of the machine. There was NO calibration for the densitomer itself. Whenever we had to change a bulb in it the procedure was to adjust a potentiometer on it until it gave a certain reading. Where the information on what this reading really was I was never able to find out. neither was I ever able to find out where the conversion graph came from. Suffice it to say that we used this system despite many changes in production batches of fim badges, changes in the chemicals used for developing, etc. without any recalibration of the equipment. The curve on the graph paper only covered a fairly narrow range of the densities measureable by the densitomer. In some cases the film badges were so dark that the optical density reading obtained was completely off the graph. The graph was already non-linear but despite this, when this problem occurred we would just guess a value and record this. This state of affairs continued until after I discovered the Cobalt 60 at Taddje. Using various other sources available we were able to estimate the strength of one of the Cobalt pellets. This was then placed in a jury-rigged arrangement of laboratory stands in one of the compounds with an array of film badges at various distances from the source. By this method we were able to relate radiation received to the optical densitomer readings and thus produce a new calibration curve.

THYROID IODINE 131 MEASUREMENTS

While I was with the L.R.D.U. group at Mt. Clarence, part of our [illegible]tion was to collect thyroid glands from sheep in what was thought to be the general fallout path. Several of the L.R.D.U. members had in fact had an additional 1 weeks training in Melbourne following the 2 week general course which we all undertook. At tat time in central Australia there had in fact been a drought for about 2 years in places. In some instances sheep farmers were paying to have dying sheep removed from their property so that they did not foul the few remaining sources of water. Imagine their delight when we came along and offered them $10 (5 pounds) for the thyroid glands of just on sheep. In many cases the farmer would choose the sheep closest to death anyway. The point of the matter is that the project was based on the assumption that the sheep would have been grazing on grass and thus would have ingested any Iodine 131 which may have fallen. If there is no grass to eat then no matter how much Iodine 131 may have fallen there will be no sign of it in the thyroid glands. My belief is that the project may have been dreamed up by U.K. scientists who had absolutely no idea of the Australian outback. I also believe that the only accurate readings of Iodine 131 that may have been obtained was when the fallout from one of the earlier tests went south-east towards Adelaide instead of north-east and thus fell on reasonable pastures where sheep were in fact grazing. These measurements unfortunately were not done by Maralinga scientists and were thus discounted.

FILM DOSIMETERS.

Quartz fibre dosimeters were used in the forward area with the [illegible] of being able to get instantaneous readings of radiation received. Two different types of instruments were in use, one with a 10 times higher range than the other. If my memory serves me correct I believe that one may have been 0-50 and the other 0-500. In the the post-test phase when radiation levels had reduced somewhat only the 0-50 instruments were supposed to be issued but because of the limited number of them available quite often 0-500 instruments were issued which would not have registered anything. The quartz fibre dosimeters in use had a fragile quartz fibre assembly and microscope optical assembly to read it with. Because of the fragile nature of them many of them were in a damaged condition e.g. either the fibre could not be seen at all, or there might be no movement of the fibre between the charged and discharged states. In many cases I found that some of the personnel working in the forward area would demand a dosimeter even though no workable ones were available because many of them were under the mistaken impression that the wearing of them prevented on from receiving any radiation. The dosimeters were electrically charged by means of a battery operated charging unit. The majority of the charging units were defunct because of different electronic faults, and it was difficult to obtain good batteries for the remainder. A large chest freezer unit was kept at the RB unit and this contained hundreds of batteries of different types. Most of the batteries were past their expiry date and had suffered water damage. The general procedure was to grab a handful of the batteries for the dosimeter chargers and hope that amongst them one could find one that still had enough charge to operate. In many cases the radiation levels recorded for persons working in the forward was 'guesstimated' based on the known levels in the general area where they were supposed to be working, and perhaps correlated with the readings of someone who might have been lucky to actually have a working dosimeter.

HOT SPOTS.

While at Mt. Clarence with the L.R.D.U. during the actual Antler tests I was in charge of a small group whose duty was to drive to the expected path of the fallout and take measurements as the fallout passed over. In some cases the wind changed and we were nowhere near where the fallout actually fell. In other cases we were directly underneath. In fact in one case I received fallout in my hair which even after about 3 washings could not be removed so the camp barber just gave me a crew cut which solved the problem. In a couple of instances while we actually in place and waiting for the fallout to arrive I decided on my own initiative to do some general background readings using a G.M. counter. Imagine my suprise when I found occasional spots where the count was already about 100 times greater that the general background. After some investigation I established that this was usually associated with clumps of spinifex, and not on the general cleared areas. My assumption was that it must have been the results of fallout from an earlier series of tests, perhaps the Buffalo series, and that wind action might have resulted in the concentrating of the actual fallout material in the vicinity of the spinifex plants. An alternative possibility occurred to me much later. It was noticed that about a year after the tests by which time there had been some rain, that when viewed from the air there was a definite increase in the vegetation in the areas where fallout had actually occurred. This was explained to me by one person who pointed out that the whole area was known to be deficient in rare earths often necessary for plant growth and that perhaps the fallout was in fact providing the necessary rare earths. Suprisingly enough this fact was bought to my attention again only about 6 years ago when I was working with a scientist whose Ph.D. thesis in botany was based in the central Australian region. In his thesis he drew attention to certain clumping of vegetation which he had observed in the area. He was not aware that atomic fallout had been over those areas and was quite suprised when I bought it to his attention. He agreed that this was the only logical explanation which satisfied his observations. This would tend to suggest then that even 20 years after the event the affect of atomic fallout in central Australia can still be observed.

DOCUMENT

GROUP

SEPARATOR SHEET

RC 144

[redacted]

FILE NO. [redacted]

8 June, 1977

[redacted]
Australian Radiation Laboratory,
36 Lonsdale Street,
MELBOURNE, VIC. 3000.

[redacted]

I am writing to you hoping you may be able to help me obtain either radiation records or medical records on [redacted] who was employed with the Atomic Weapons Testing, Health Physics Branch at Maralinga for the period 25 July 1957 to 22 June 1959.

In 1968 [redacted] entered the Royal Brisbane Hospital and while there his white cell count was found to be lower than normal. The only cause the doctors could attribute this to was the work he was doing in 1957 to 1959.

In January this year, [redacted] was again hospitalized with suspected Thypoid. The doctors' initial diagnosis was supported by a low white cell count. However further tests were conducted and it was found that [redacted] was suffering from Salmonella gastroenteritis.

As the doctors are concerned about his low white cell count he has been undergoing a series of tests in Townsville including X-rays, and pathological tests to try to determine the reasons for the low white cell count.

[redacted] is of the opinion that during his work with you he was exposed to a higher level of radiation then was considered safe. This overexposure is now causing problems with his health mainly the low white cell count.

Should this be correct I feel that he may be entitled to compensation even though the accident occurred some 20 years ago.

...2/

3

I have written to Weapons Research Establishment for any radiation or medical records that they might have retained. However their only records were the standard Commonwealth Public Service X Ray and medical examination for appointment. This medical examination normally does not include blood tests and as such their records would show that he was suitable for employment. These examinations were conducted in April 1964 and January 1966 prior to his hospitalization in 1968. There are no records for the period 1957-1959.

Because of the nature of his work with you he is unable to inform me of any instances when he may have been overexposed to radiation and it is in this regard that I am writing to you. As it possible to obtain some form of documentary proof that he was at some time over-exposed to rddiation, which may be used in a case for compensation. I fully realize such proof may be difficult to obtain but any assistance that you could give would be appreciated.

Yours sincerely,

[redacted]

[redacted]

DEPARTMENT OF DEFENCE

WEAPONS RESEARCH ESTABLISHMENT

SALISBURY, SOUTH AUSTRALIA • BOX 2151, G.P.O., ADELAIDE, S.A. 5001 • TELEGRAMS: "WEAPONS" ADELAIDE • TELEPHONE 2599111

ther information refer to [redacted] Ext 487 In Reply Quote SP28/5/10

17 FEB 1977

RECEIVED
AUSTRALIAN INSTITUTE OF MARINE SCIENCE
23 FEB 1977

Secretary,
Australian Institute of Marine Science,
P.O. Box 1104,
TOWNSVILLE, QLD. 4810

Attention: Personnel Officer

[redacted]

I refer to your menorandum J.F.J.:D.M.B. of 2nd February, 1977.

2. [redacted] was employed as a Temporary Technical Assistant Grade 1 with this Establishment at Maralinga from 25th July, 1957 to 22nd June, 1959, when he left by way of resignation. We have no medical records in respect of that period of employment.

3. However, he was re-employed by us as a Temporary Technical Assistant Grade 2 on 6th April, 1964, at our Woomera installation. His medical and X-Ray examinations in respect of that engagement were satisfactory.

4. On 11th January, 1966, [redacted] was Permanently Appointed under the Public Service Act and was accepted as a contributor to the Superannuation Fund - indicating satisfactory medical and X-Ray standards. He subsequently tendered his resignation and from close of business on 29th March, 1967.

5. Sick Leave reccrds maintained at this Establishment in respect of [redacted] from 1964 to 1967 show absences totalling 1 week 5 hours 45 minutes for illnesses of only a minor nature.

[redacted]

xxxxxx AA47165

JFJ:DMB

2 February, 1977.

Chief Administrative Officer,
Weapons Research Establishment,
G.P.O. Box 2151,
ADELAIDE. S.A. 5001

[redacted]

[redacted]

Reference A: Our memorandum dated 15 July, 1976.
B: Your SP28/5/10 dated 26 July, 1976.

It would be appreciated if you could forward any records either medical or radiation that you might have on the abovenamed officer for the period 1957/58. During this time [redacted] was employed with Department of Supply, Atomic Weapons Testing, Health Physics Branch, under [redacted].

The reason for this request is that in 1968 while undergoing treatment for another illness, doctors at the Royal Brisbane Hospital found that [redacted] has a lower than normal white cell count and after extensive testing considered that this condition may have been attributed to his work with the Health Physics Branch in 1957/58. Recently after being hospitalized with severe food poisoning, doctors at the Prince Henry Hospital, Sydney, found that his white blood cell count was still lower than normal.

[redacted] has requested this information for a possible compensation claim and in case he is ever hospitalized the information may be of assistance to his doctors.

[redacted]

The following information on [redacted] employment has been found in Health Physics files held by the Department of National Development and Energy.

[redacted] attended a course of lectures and demonstrations in Health Physics conducted by the Commonwealth X Ray and Radiation Laboratories in Melbourne, from 25 July to 9 August 1957, as a member of the Australian Radiation Detection Unit. (ARDU). From mid-August 1957 to October 1957 he was at Mt Clarence in South Australia which was the ARDU base for field observations of fallout, particularly along the Alice Springs road, following the atomic tests at Maralinga.

On completion of the Mt Clarence project, [redacted] joined the Australian Health Physics Team (AHPT) at Maralinga on 11 October 1957. He was initially rostered for a 6 month tour of duty at Maralinga but remained with AHPT until the end of June 1959 when he left to take up an appointment at the [redacted] During his time at Maralinga the Australian Health Physics Representative on the Range was [redacted] It is recorded that [redacted] was the sole Health Physics officer on the Range over the Christmas period 23/12/57 to 4/1/58.

The Australian Health Physics Team stationed at Maralinga provided support for the Australian Health Physics Representative who was responsible for the day-to-day supervision of radiological safety on the Range in the inter-trials periods. Duties of AHPT included :

- control of personnel entering radioactive areas, including briefing, radiation monitoring and decontamination
- maintenance of measuring equipment
- water and air sampling
- delineation of contaminated areas
- radiation surveys of test sites
- organisation of Services training courses in radiation detection.

During trials operations, radiological safety control was exercised by specialist teams from the [redacted] in these periods, the AHPT did not operate as a separate entity but rather as individuals attached to the various U.K. health physics groups.

Personnel involved in 1956 and later test programs at Maralinga were subject to radiological safety regulations approved by the U.K. authorities and the Australian Weapons Test Safety Committee. The maximum permissible doses under these regulations were based on the recommendations of the International Commission on Radiological Protection (ICRP) and were subject to the proviso that every endeavour should

s made to keep the average [illegible]

ICRP recommendations on the maximum permissible ose for irradiation of blood-forming organs, for occupational xposure, were:

- in 1955, 0.3 roentgens per week (equivalent to 15 oentgens per year)

- from 1958, 5 rems per year (equivalent to 5 roentgens er year).

A summary of film badge records shows [redacted] eceived gamma radiation totalling 4.48 roentgens over the eriod September 1957 to June 1959.

DOCUMENT

GROUP

SEPARATOR SHEET

[redacted]

RC 144

COMPENSATION (COMMONWEALTH GOVERNMENT EMPLOYEES) ACT 1971
[redacted] - Formerly Technical Assistant
Department of Defence

In the matter of the claim of [redacted] for compensation in respect of nuclear radiation damage.

DETERMINATION

1. On the evidence, before me, including specialist medical opinion, I find that the condition of chronic mild myelofibrosis suffered by the said [redacted] constituted a disease due to the nature of his employment within the meaning of section 10 of the Commonwealth Employees' Compensation Act 1930, as amended.

2. NOW THEREFORE, in pursuance of the provisions of the Compensation (Commonwealth Government Employees) Act, including sub-sections 4(2) and 104(1) of that Act, I hereby determine :-

(a) the said [redacted] suffered a disease, namely, chronic mild myelofibrosis in circumstances under which the Department of Defence would have been liable to pay compensation under the Commonwealth Employees' Compensation Act 1930, as amended;

(b) the Department of Defence is therefore liable to pay compensation in relation to the disease in accordance with the provisions of the Compensation (Commonwealth Government Employees) Act 1971.

[redacted]

[redacted]

Delegate of the Commissioner
for Employees' Compensation.

31 MAR 1983

ROYAL NORTH SHORE HOSPITAL

CA:jmc
Your Ref: 82/3152

22nd December, 1982

The Commissioner for Employees
Compensation.
P.O. Box 196,
WODEN. ACT. 2606

Re: [redacted]

Dear Sir,

In regard to your letters of the 1st September and 3rd December, 1982, I would like to detail the case notes as I have obtained them.

[redacted] aged 43 years was referred to the Haematology outpatients clinic on the 4th August, 1982 for investigation of splenomegaly. The event which precipitated attendance at the clinic was the recent deterioration of abdominal pains which the patient had suffered intermittently for many years. However, in the past three to six weeks, the pain had been worse and on examination by [redacted] local doctor, tender splenomegaly was noted. [redacted] stated that in 1958 at the age of 17, he worked at the Maralinga atomic bomb testing sites and that he had had excessive exposure to radiation. Six months after leaving the test sites, whilst in England, the patient had a febrile illness of undetermined cause although he was told at the time that he had signs and symptoms consistent with malaria but no definitive diagnosis could be proved. In 1969 the patient was admitted to Brisbane Hospital during which time an abnormal blood count and bone marrow investigation were found. I do not have the details of these results which will be necessary for you to obtain in order to completely evaluate [redacted] case. In 1977 the patient suffered paratyphoid fever and was admitted to Prince Henry Hospital where again an abnormal bone marrow was found although a specific diagnosis was not told to [redacted] For many years, [redacted] has also complained of pains in his right foot and also of dyspnoea on minimal exertion.

Clinical examination revealed a well looking man with blood pressure 110/70, pulse 80 and regular, respiratory rate 16 per minute. Cardiorespiratory examination was normal, several small subcutaneous nodules on the trunk and limbs were noted, there was no lymphadenopathy and abdominal examination revealed a very tender xiphisternum, borderline hepatomegaly and an enlarged spleen palpable 3-4cm below the left costal margin and the spleen was tender.

Cont'd page 2.

Investigations performed were as follows:-

Blood Count. Hb 133 g/l, MCV 85, MCH 29.7, MCHC 344, Platelets 570 x 10^9/l, ESR 5mm in 1 hour, WCC 5.2 x 10^9/l, Reticulocytes 2%, Differential on the white cell count Band Forms 4%, Seg. Neuts 75%, L 15%, M 5%, E 1%.

The blood film was grossly abnormal and showed nucleated red blood cells, moderate to marked anisocytosis and poikilocytosis, and a moderate degree of polychromasia.

Bone marrow aspiration was performed and was difficult to aspirate both from the right posterior iliac crest and from the sternum. A reduced number of marrow particles were obtained and on microscopy the particles appeared dense and abnormal. Erythropoiesis was dyserythroid. Granulopoiesis was dysplastic and only occasional megakaryocytes with dysplastic features could be found. Lymphocytes were present with occasional atypical forms.

Bone marrow trephine showed an abnormal marrow with hypercellular masses of haemopoietic marrow filling the marrow space with irregular islands of fat cells between them. All three cell lines were present but megakaryocytes appeared unusually prominent, variable in size and forming clusters. A marked increase in reticulin was present and the changes were those of a myelofibrosis.

Biochemical examination was unremarkable apart from a mildly elevated creatinine of .12 and urea of 7.4mm/l.

Chest X-ray was normal.

Liver spleen scan showed a liver of normal size with even uptake and an enlarged spleen with even uptake.

A bone marrow scan with technician 99 sulphurcolloid was performed and showed minimal uptake of sulphurcolloid in the sacrum and pelvis and no uptake in the sternum with no evidence of peripheral extension into the long bones.

Interpretation was of marrow hypoplasia.

In view of the dyspnoea, respiratory function tests were performed and showed mildly abnormal alveoloarterial oxygen difference, normal gas transfer, normal lung volumes, and normal airways resistance with normal spirometry and no significant upper airways obstruction. However, an exercise test was also attempted in order to pick up early degrees of pulmonary fibrosis. This test was aborted because of severe hyperventilation causing dizziness and distress therefore making the measurements of gas transfer questionable.

Biopsy of the skin lumps was performed and showed benign lipomata.

In summary, [redacted] appears to have marrow hypoplasia with marrow fibrosis and splenomegaly which in our opinion, is enlarged due to extra medullary haemopoiesis. I have discussed [redacted] case with [redacted] staff haematologist, who has also seen [redacted] in consultation and we agree that [redacted] is a most unusual case. In attempting to give an informed opinion on a probable cause of the condition which [redacted] has, I have tried to research previous cases of radiation exposure and various articles and books as referenced below. Unfortunately although there is a lot of literature on the effects of radiation, much of this has been directed to the effects of low level ionising radiation and the effects of acute high dose radiation exposure in various reactor site accidents. Historically it would appear that [redacted] exposure was more subacute and therefore one might expect some differences in his response to the postulated excessive radiation exposure. From examination of the literature both human and animal studies and also from the knowledge obtained from patients treated therapeutically with radiation, it is known that long term effects of marrow irradiation may result in fibrosis with marrow hypoplasia. Whether or not such marrow hypoplasia if generalised would then result in the formation of extramedullary haemopoiesis such as in the spleen, I cannot answer although theoretically this is possible and in fact known to occur in some myeloproliferative disorders of the marrow. I plan to perform isotopic studies with iron 59 and by means of counting over the spleen and other organs, we can determine if in fact [redacted] has extramedullary haemopoiesis in the spleen. As I have indicated above, it would be helpful if the original reports from Brisbane and also from Prince Henry Hospital could be obtained to see if there was also marrow fibrosis or hypoplasia noted at those times. The opinion of [redacted] and myself is that [redacted] probably has radiation induced marrow fibrosis. If such is the case, then the prognosis is difficult to determine. If the patients marrow function remains at its present level and I believe it will, the patient should not come to any harm from marrow failure. The presence of splenomegaly poses some slight risk of rupture especially if the patient is subjected to any trauma and the great danger would be if the patient needed to have a splenectomy, as we believe that most of the patients marrow function, is coming from his spleen. The increased incidence of leukaemia has been reported many times in the literature following radiation exposure which is probably best documented in the survivors of the Nagasaki Hiroshima atomic bomb explosion. Although I am unable to give the likelihood of leukaemia developing in [redacted] it is our opinion that it is certainly higher than in the normal population and if such a leukaemia does develop, it is often very refractory to normal therapeutic measures. I am uncertain as to the cause of the patients respiratory complaints and it may be necessary to attempt a repeat of his respiratory function test in the future, however, I am suspicious that there may be early degrees of pulmonary fibrosis which may progress in future years.

Cont'd page 4.

It is known in animal and human studies of radiation exposure that often the end result is replacement of the affected organs by fibrotic tissue and in [redacted] case, the ultimate prognosis would depend on the degree to which this occurs in the various organs such as the lung, bone marrow and blood vessels. In short, [redacted] prognosis is that he is likely to have a shortened life span from the effects of his disease although I can be no more definite that this.

Treatment at this stage consists mainly of pain relief as required and avoidance of any situations likely to lead to traumatic injury of the spleen. Monitoring of the patient's blood count and possibly bone marrow at regular intervals say 6 to 12 monthly will be recommended to [redacted] to look for deterioration of his condition.

Please find enclosed copies of the patients medical reports and clinical notes.

Please do not hesitate to contact me again if you need further information or clarification regarding [redacted] condition.

[redacted]

Encl:

Refs:

[redacted] Studies in the Pathology of Radiation Disease 1965 Pergamon

Medical Research Council. The Hazards to Man of Nuclear and Allied Radiations. A Second Report to the Medical Research Council. Dec 1960
Her Majesty's Stationary Office, London.

[redacted]
The Medical Basis for Radiation Accident Preparedness. Elevier Nth. Holland Inc., 1980

COMPENSATION (COMMONWEALTH GOVERNMENT EMPLOYEES) ACT 1971
[redacted] - Formerly Technical Assistant
Department of Defence

In the matter of the claim of [redacted] for compensation in respect of nuclear radiation damage.

DETERMINATION

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(b) the Department of Defence is therefore liable to pay compensation in relation to the disease in accordance with the provisions of the Compensation (Commonwealth Government Employees) Act 1971.

[redacted]

[redacted]

Delegate of the Commissioner for Employees' Compensation.

31 MAR 1983

DOCUMENT

GROUP

SEPARATOR SHEET

HARVEY · JOHNS · OWENS · ROSS

The Principles and Practice of Medicine

Eighteenth Edition

APPLETON-CENTURY-CROFTS / New York
A Publishing Division of Prentice-Hall, Inc.

fibrosis also occurs more frequently in P^{32} treated patients.[89] Alkylating agents, such as busulfan or mechlorethamine, and other cytoxic agents, such as 6-mercaptopurine and demecolcine, have been used to inhibit the proliferation of marrow elements with some degree of success.

MYELOID METAPLASIA WITH MYELOFIBROSIS. Many descriptive terms have been applied to the group of illnesses characterized by a leukoerythroblastic anemia, enlargement of the spleen and liver as the result of myeloid metaplasia, and a patchy or generalized fibrosis of the bone marrow. The etiology of this syndrome remains obscure. There is an increased incidence of myeloid metaplasia among atom bomb survivors and others exposed to ionizing radiation.[89] One opinion relates this disorder to a leukemia of reticuloendothelial cells involving principally the marrow, spleen, and liver. At present, many group this condition with the myeloproliferative disorders largely because of their overlapping clinical features and because of the occasional transition from one form to another.[6, 90]

Clinical Presentation. Myeloid metaplasia, a rather uncommon illness, occurs in both sexes with equal frequency, generally after age 50. The primary complaints are weakness, easy fatigability, abdominal discomfort, and aching in the extremities, especially the legs. Less frequent symptoms are hemorrhage (skin and gastrointestinal tract), gout, and weight loss. Common physical abnormalities include pallor, ecchymoses or petechiae, striking splenomegaly, and more modest hepatomegaly. Icterus is present at times.

Although a few patients may have a normal or even increased red blood cell mass early in their illness, one of the most characteristic features of this disorder is the leukoerythroblastic anemia. The red cells show marked anisocytosis and poikilocytosis. Nucleated red cells are often seen. The reticulocyte count is normal or moderately elevated.

The white cell count is usually elevated, and the differential shows a shift to the left. All types of immature myelocytes may be encountered in the peripheral blood, including an occasional myeloblast. The alkaline phosphatase stain shows high or normal amounts of enzyme; rarely, low values are obtained. In most cases the platelet count is elevated and large, odd shapes are often encountered.

Attempts to secure marrow by aspiration may be unsuccessful. Biopsy techniques provide the means of demonstrating the myelofibrosis and hypocellularity present. Osteosclerosis may be seen also. These marrow changes may be patchy early in the disorder; later they may be generalized. Indeed, it is not uncommon to encounter foci of hematopoietic-cell hyperplasia. Biopsy or aspiration preparations from the spleen, the liver, and, rarely, the lymph nodes may show extensive extramedullary hematopoiesis (myeloid metaplasia).

X-rays of the bones in 25 to 50 percent of cases show a patchy irregular osteosclerosis. The cortex of the long bones is often thickened. Osteoporosis has also been noted. The blood uric acid level is elevated in about 50 percent of instances, at times to twice normal levels.

Diagnostic Considerations. The hallmarks of myeloid metaplasia with myeolofibrosis include a leukoerythroblastic anemia, marked splenomegaly and hepatomegaly with prominent myeloid metaplasia, and fibrosis or sclerosis of the bone marrow.

The diagnostic problems commonly confronted stem in part from the transition forms which exist between polycythemia vera, chronic myelocytic leukemia, and this disorder. In other instances, myeloid metaplasia must be distinguished from an aplastic or hypoplastic anemia. In addition, the physician must remain alert to the several possible causes of leukoerythroblastosis.

"Leukoerythroblastosis" is a term used to describe the presence of nucleated red cells and various immature myelocytic forms in the peripheral blood. When severe, this abnormality is striking, persistent, and usually accompanied by anemia. In its mildest forms there may be no anemia and only a few transiently circulating abnormal cells. Leu-

TABLE 2: Causes of Leukoerythroblastosis

I. Due to Abnormal Myeloproliferation Following
   A. Blood loss or hemolysis
   B. Nutritional deficiency anemias

II. Due to Myelophthisic Disorders
   A. Tuberculosis, especially disseminated
   B. Carcinomatosis (lung, breast, prostate)
   C. Xanthomatosis (Gaucher's disease and others)
   D. Lymphoma
   E. Myeloma
   F. Myeloproliferative disorders

III. Due to Severe Illness, Stress, Agonal States
   A. Infection
   B. Heart failure
   C. Uremia

koerythroblastosis may result from myeloproliferation or from myelophthisic disorders (Table 2). Carcinomatosis and other myelophthisic processes are the most frequently encountered causes of marked leukoerythroblastosis.

Clinical Course and Therapeutic Management. The average prognosis for life ranges from four to five years, but with good supportive care many patients live much longer. The main continuing clinical problems are those stemming from the anemia, the markedly enlarged spleen, or the hemorrhagic episodes.[6, 90, 93]

Symptoms resulting from severe anemia are relieved by transfusion. Androgens have improved the anemia in a few instances. Not infrequently, a hemolytic anemia develops, and the Coombs test may rarely become positive. Adrenal corticosteroids have benefited these individuals as well as a few others. Splenectomy has been performed to relieve this hemolytic problem. Thrombocytopenia with bleeding also develops on occasion and may be relieved by splenectomy. In these instances splenectomy has been performed de-

TABLE 3: Contrasting Clinical Features of the Chronic Myeloproliferative Disorders

CLINICAL FEATURES	POLYCYTHEMIA VERA	MYELOID METAPLASIA WITH MYELOFIBROSIS	CHRONIC MYELOCYTIC LEUKEMIA
Presentation	50% of cases	0	0
Pruritis	Plethora; erythema	0	Leukemic infiltrates
Skin lesions	When reduced hemoglobin exceeds 5 g per 100 ml	0	0
Ruddy cyanosis	Rare	Occasional	Rare
Jaundice	25–50% of cases	10–20% of cases	Occasional
Thromboses	Epistaxes, skin, central nervous system, gastrointestinal and joint bleeding	Especially gastrointestinal and skin bleeding	
Hemorrhage	50% of cases	Sometimes early	Occasional
Hypertension	Modest, 30–50% of cases	Most cases often large	Modest
Hepatomegaly	Usual, often large	Often huge	Large
Splenomegaly	May be absent early	10–20% of cases	Occasional
Peptic ulcer	10–20% of cases	5–10% of cases	Rare
Gout	Approx. 5% of cases	Approx. 20% of cases	Occasional
Bone pain	Frequent	++	+++
Sternal tenderness	0		50–75% of cases
Course			
Average prognosis	10–20 years	4–5 years or more	3–4 years
Incidence of acute leukemia	1–10% of cases Related to P^{32} therapy	0	Terminal acute blastic crisis usual
Terminal events	Hemorrhage Thromboembolism Cardiovascular disorder Transition to myelofibrosis or chronic leukemia	Hemorrhage Thromboembolism Infection	Hemorrhage Infection

spite the fear attending removal of large amounts of hematopoietic tissue and the difficult operative and postoperative problems.[93] Following splenectomy, the liver enlarges progressively (myeloid metaplasia), and the general course of the disease continues.

External radiation has been directed at the spleen to relieve local symptomatology or to combat hemolytic anemia or thrombocytopenia. The potential danger of irradiating the areas of extramedullary hematopoiesis has been stressed also. Busulfan has induced a decrease in spleen size in some patients and an improvement in their hematologic status, but it may have adverse effects on the remaining hematopoietic tissue.

The immediate causes of death relate to underlying cardiovascular disease affected adversely by anemia, thromboembolic events, or hemorrhage. A transition to chronic myelocytic or acute myeloblastic leukemia may take place before death.

CONTRASTING CLINICAL FEATURES OF THE CHRONIC MYELOPROLIFERATIVE DISORDERS. Polycythemia vera, myeloid metaplasia with myelofibrosis, and chronic myelocytic leukemia are considered together primarily because they manifest a chronic uncontrolled proliferation of hematopoietic cells and because their clinical features often overlap or change in an interrelating fashion with the passage of time. Although certain basic morphologic resemblances are stressed, no implication with respect to etiology is in-

TABLE 4. Contrasting Hematologic Features of the Chronic Myeloproliferative Disorders

LABORATORY EXAMINATION	POLYCYTHEMIA VERA	MYELOID METAPLASIA WITH MYELOFIBROSIS	CHRONIC MYELOCYTIC LEUKEMIA
Hematocrit	Increased, often > 60	Decreased May show increase early	Decreased May be near normal early
Hemolysis	Normal erythrocyte survival	Occasional, with positive Coombs test	Normal erythrocyte survival
White Blood Cell Count	Usually 10–50,000; rarely > 100,000	Usually 20–50,000; rarely > 100,000	Generally elevated; often > 100,000
Differential	Normocytic red cells Occasional nucleated red cells Occasional myelocyte	Bizarre red cell shapes Frequent nucleated red cells Frequent myelocytes, occasional myeloblast	Normocytic red cells Occasional nucleated red cells Orderly shift to left with myelocytes, occasional myeloblast
Platelets	Usually elevated; > 400,000 in 50% of cases	Marked elevation, often bizarre forms May develop thrombocytopenic purpura	Usually elevated
Granulocyte Alkaline Phosphatase	Highest values	High to normal	Low to absent
Ph' Chromosome	0	0	+
Bone Marrow	Cellular Hyperplasia of all elements Read as normal in 50% of cases	Fibrosis and osteosclerosis Reticulum cell increase Patchy lesions early Focal areas of increased cellularity early	Myeloid hyperplasia At times increased red cells and megakaryocytes or patches of fibrosis
Myeloid Metaplasia	Rare foci in liver, spleen	Marked in spleen, liver At times in lymph nodes	0 (Myeloid infiltrates)
Blood (RBC) Volume	Increased	Normal (anemic)	Normal (anemic)
Blood Viscosity	Increased	Normal	Normal
Uric Acid	Increased	Increased in 50% of cases, at times twice normal	Increased
Immunoglobulins	Rare monoclonal gammopathy	Rare increase in γM rheumatoid factor	—
X-rays of Bones	Usually normal	Patchy or diffuse osteosclerosis, thickened cortex, or osteoporosis in 25–50% of cases	Occasional subperiosteal new bone, osteolytic lesions, transverse lines at end of long bones

fibrosis as well as myeloid metaplasia in the spleen, although more frequently tuberculosis seems a secondary occurrence. However, present data do not permit definite conclusions concerning these or other suggested causal agents. The common proliferative abnormality seems neoplastic in character.[6, 89, 90]

The contrasting clinical features of these chronic myeloproliferative disorders are presented in Tables 3 and 4. Continuing clinical experience serves to emphasize the diversity of findings manifested by different patients and by the same patient during the course of his disease. A patient with classic polycythemia vera may after many months develop an increasing anemia and progressive enlargement of the spleen and liver. Appropriate biopsies may reveal myelofibrosis and extramedullary hematopoiesis associated with a leukoerythroblastic anemia. Similarly, an individual with polycythemia vera may develop all the features of chronic myelocytic leukemia. This may evolve directly from the polycythemic status or may occur as a blast crisis in a person whose illness had previously become chronic myelocytic leukemia. The use of P^{32} therapy or other forms of radiation therapy has been linked to a tenfold increase in the incidence of acute leukemia.[89, 94]

Some patients with clear-cut myeloid metaplasia with myelofibrosis show transient polycythemia early in the course of their disease. The myeloid metaplasia syndrome seems unique among the myeloproliferative disorders because of the occasional associated occurrence of an autoimmune hemolytic anemia and the occasional associated development of thrombocytopenic purpura unrelated to therapy. A distinctive relationship appears to exist between the Ph' chromosome and chronic myelocytic leukemia, although exceptions have been reported.[6, 90]

59 The Lymphoreticular Proliferative Disorders

Lymphoid and reticuloendothelial tissue is located in many areas of the body. Apparent excessive production of lymphocytes, plasma cells, monocytes, and reticulum cells is a feature of several regional or systemic diseases. Although our current understanding of the homeostatic mechanisms regulating the basal rates of cell proliferation is limited, it seems clear that an increased production follows a variety of exogenous and endogenous stimuli and is related to the resulting immune responses. As with the myeloid disorders, the proliferative abnormalities of the lymphoreticular cells may be categorized further with respect to 1) their neoplastic or nonneoplastic characteristics, 2) the cell type(s) involved, 3) the duration of their course (acute or chronic), and 4) the associated immunologic response(s).[1, 6, 87, 88]

NONNEOPLASTIC LYMPHORETICULAR PROLIFERATION. Table 1 summarizes the variable immunoproliferative phenomena attending the majority of infectious diseases. In general, the mononuclear cell response occurs promptly after antigenic stimulation and subsides on removal of the stimulus. In acute bacterial (pyogenic) infections, lymphoreticular hyperplasia and plasmacytosis may follow the initial granulocyte response, whereas in most viral infections it is the primary event. The evidence of the associated immunologic responses (e.g., antibody titer, delayed cuta-