SCIENTIFIC BULLETINS

of the

Office of the Air Surgeon

Headquarters Army Air Forces
Washington, D. C.

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SUMMARY REPORT NO. I

STATUS OF INVESTIGATIONS OF DECOMPRESSION SICKNESS

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Summary reports reviewing the more significant findings contained in reports from service and civilian laboratories in the United States and Allied Countries are issued from time to time. It is the purpose of these summaries to make available to flight surgeons and research personnel a brief survey of the information relating to a certain subject contained in the numerous and often voluminous reports in the files of the Air Surgeon. There is no assurance that the files are complete in every detail, but it may be assumed that the summaries will fairly indicate the trend of work and opinions in the field. The information here reported is a review of the available reports and does not necessarily reflect the opinion of the Air Surgeon or his staff.

C-509-1, AF

The importance of the problem from the military standpoint can be seen from a consideration of the incidence of disqualifying symptoms among aviation personnel. About 25% of 48 R.A.F. pilots tested for a half hour at 37,000 feet were incapacited (26) and about 25% were entirely unaffected. Later studies showed the incidence of severe symptoms at 40,000 feet to be about 10% during the first hour. The test was subsequently changed to four hours at 35,000 feet. About 30% of tested men were disqualified by this procedure (28). In selecting crews for the Boeing Fortresses 36% of tested men were rejected (29). This early experience in England has been repeated recently in the United States. About 24% of five hundred men tested at Pensacola (18) had pain in some joint at the end of one hour at 35,000 feet. Of these only five percent were considered to be incapacitated at the end of the test. In the four hour test of 723 cadets at Randolph Field some 283 men (37%) failed at 38,000 feet (21). In another group of 43 cadets (19) a single test of four hours at 35,000 feet produced severe symptoms in 26%. The average duration of flight among these susceptible men was 2 hours and 33 minutes on this occasion.

Figures based on a single test do not represent the incidence of incapacitating symptoms to be expected among unselected personnel on repeated flights. Matthews noted the extreme variation in susceptability of an individual from day to day (25). For this reason, a single test does not show the true percentage of susceptible men. Indeed Matthews believed that no one was really immune to decompression sickness (25). When repeated tests are used to classify subjects the number of apparently immune people decreases. Thus a series of five tests of four hours each at 35,000 feet showed only 44% of tested cadets to be consistently free of serious symptoms (19).

These facts clearly demand some method for reducing the incidence of incapacitating pain caused by discompression. One procedure, depending upon the elimination of nitrogen from the body, has shown variable degrees of success (27,30,5), but has been considered to be too time consuming for operational purposes (30). However, recent experiences with this method shows that breathing oxygen for one hour while doing mild exercise, affords a marked degree of protection against decompression pain. Such a procedure should be useful whenever circumstances permit its use. It is of course important to the success of the method that pure oxygen be breathed at all times after beginning the denitrogenation. A modified procedure calls for breathing oxygen for twenty minutes, while exercising more violently. Subsequently the men rest a few minutes and then ascend breathing oxygen at all times. The effectiveness of this modified procedure has been only partially evaluated. A third method of prevention proposed by Professor Bazett depends upon the slow rate of re-saturation of tissues with nitrogen. Thus, the men eliminate all their tissue nitrogen gas by breathing oxygen during sleep. When they arise in the morning, they again breathe air. The protection against decompression sickness lasts for several hours because the nitrogen passes into the body slowly. This method too needs further testing. The only other important measure for the control of decompression sickness is based upon selection of immune personnel by testing in a low pressure chamber.

This method is reported to have been successful in eliminating "bends" as a problem in the Photographic Reconnaissance Units of the R.A.F.(28). However, in recent studies considerable difficulty has been encountered in devising an adequate measure of man's susceptibility to incapacitating symptoms.

The susceptibility of an individual to these symptoms is stated in terms of maximum equivalent altitude attained and duration of stay at this altitude. As mentioned above, the time he can remain at altitude is frequently limited by the development of a certain degree of joint pain since this is generally the first of the incapacitating symptoms developed. (2,4,18). One observer recommends the assessment of degree of pain according to four standards:

1. Just barely perceptible.
2. Definitely present and annoying, but not sufficient to interfere with completion of the mission.
3. Approaching the limit of tolerance, but bearable for a few minutes.
4. Unbearable, there demanding the immediate termination of the experiment.

In Evelyn's investigations on fifty medical students, each man was decompressed eight times on alternate days (2). The subject passed the test if he did not experience a grade 1 pain on more than half the trials and never experienced more than a grade 2 pain at any time. On this basis, 20% of his men could not go to 30,000 feet for four hours, 50% could, and 30% could also go to 40,000 feet for fifteen minutes. In these experiments, both the altitude and the duration of stay were found to be important in producing decompression pain. But the rate of climb had no influence on the development of joint pains. Even stepwise ascent with equilibration at several intermediate levels did not significantly increase the time that a subject could remain at 30,000 feet without symptoms. This investigation thus defined the important variables to be considered in assessing tolerance to decompression. The method is too long to be used routinely.

Data on 500 cadets obtained in the course of indoctrination at Pensacola (18) has been analyzed. These men were tested at 35,000 feet for about one hour. Skin symptoms were noted in 15 percent of these men and occured early in the test. Joint pains, however, usually occured late and affected 24% of the subjects. Only 5% of these had incapacitating pain. The incidence of "chokes" was very low, about 0.8%. While such short single tests are useful for indoctrination purposes they are not entirely suitable for classification because of the variability of individual tolerance from day to day.

A group of subjects tested at Wright Field were taken to 20,000 feet at 4,000 feet per minute, then to 30,000 feet at 2,000 feet per minute, and finally to 40,000 feet at 1,000 feet per minute (5). About 20% of the men subjected to this routine had no pain even after 120 minutes at 40,000 feet. The other men developed pain after an average stay of 66 minutes at 40,000 feet equivalent altitude.

The authors emphasize the extreme variability in susceptibility among individuals as well as daily variability in the reactions of an individual. They reported that the incidence of symptoms was greater in the morning than in the afternoon. Further research on the influence of sleep, time of day, and blood glucose level was recommended.

In another group of ninety-one subjects tested at Wright Field in a total of 396 decompressions, only 1.6% developed incapacitating symptoms below 30,000 feet (6). This incidence was considered to be too low to warrant any preventive measures. However, the incidence of symptoms during decompression to equivalent altitudes above 30,000 feet was high enough (ca 6%) to demand some preventive measures.

In a more recent study, 43 cadets were subjected to five decompressions to an equivalent altitude of 35,000 feet for four hours (19). "Bends" and "chokes" were the most frequent cause of disability and either of these was employed as a valid disqualifying symptom. The percent incidence of disabling symptoms in this group varied on successive tests. Thus, 26% of the subjects would have been disqualified on the first test, 16% on the second and third, 19% on the fourth, and 29% on the fifth. A further difficulty for accurate classification was the observation that the average time the disqualified men were able to stay at 35,000 feet decreased from 2 hours and 33 minutes in the first test to 1 hour and 9 minutes in the third. This average time interval then remained unchanged on the fourth and fifth tests.

Further analysis (20) shows that a single hour test at 35,000 feet is inadequate for classification. Thus in a group of subjects, classified by a series of five tests, it was shown that 30% of ineligible men would have been qualified on the basis of the first test and 7% of eligible subjects would have been rejected. To improve the classification, a series of three tests for each man was recommended. This would provide a measure of the degree of susceptibility in terms of the number of times a man fails out of three attempts.

As an alternative procedure to repeated tests, a single decompression at 38,000 feet has been suggested (23). At this altitude, the onset of symptoms is earlier than at 35,000 feet. Furthermore, all the susceptible men seem to be eliminated in three hours since the rate of elimination of subjects falls to very low values at this time.

In accordance with these findings the incidence of incapacitating symptoms of decompression sickness is significant only at altitudes above 30,000 feet (2, 6). Furthermore, the rate of climb (up to 4,000 feet per minute) has no significant effect on the incidence of serious symptoms (2, 21). The physiological stress is, however, measured by the altitude attained and the time at that altitude (2,5,6,21). The relation between these two variables is such that the higher the altitudes, the shorter the average time before onset of incapacitating symptoms. In addition, the higher the altitude the greater is the percent incidence of symptoms in a population (21,23). Thus, men differ in susceptibility in two respects, the altitude they can tolerate and the time they are free of symptoms at altitude. Both measures of susceptibility are of importance unless sufficient numbers of essentially immune men can be found.

The principal difficulty in selecting immune personnel is the variability in a subject's reactions to decompression (2, 4, 19). The time a susceptible man is free of symptoms at a given altitude is extremely variable (19). In principle, the susceptibility of the individual at a particular altitude is best expressed as the probability of the occurrence of pain after a certain time. To evaluate this probability requires several tests on each cadet and is, therefore, a time consuming procedure.

Despite the difficulty of accurate classification a single test is not useless. Thus a statistical analysis of results obtained in Canada (21) was made to determine the probability that one man in a crew of five would be incapacitated on a mission at 35,000 feet. This event was certain after about 1 hour and 15 minutes if the crew was unselected. If selected by one test the corresponding time was 2 hours. When the men were classified on the basis of four tests in a decompression chamber there was a fifty-fifty chance that no member would be afflicted at the end of four hours. The problem of selecting large numbers of crews free of symptons was considered impractical (21). All that can be done by one or more tests is to reduce the probability of occurrence of serious symptoms in the crew. The other side of the picture, of course, is the false rejection of relatively immune men when a single test is employed. However, this is not a complete waste of men for there is still plenty of flying to be done below 30,000 feet.

In view of the difficulties of selecting relatively immune personnel further studies of methods of preventing decompression sickness should not be neglected. The joint pains have usually been attributed to formation of gas bubbles in the capsular fluids. According to this view the bubbles occur because the tissues are super-saturated with nitrogen. Therefore, preliminary denitrogenation has been tried as a means for preventing incapacitating joint pains and other symptoms arising from decompression. To accomplish the denitrogenation, the subject breathes pure oxygen and exercises for an hour or more before being decompressed. At Wright Field, it was found that exercise alone did no good and oxygen alone was not very effective (5). However, preliminary exercise while breathing oxygen did significantly delay the onset of pain at equivalent altitudes of 40,000 feet. Thus, a group of twenty men in which symptoms developed on the average after 58 minutes at 40,000 feet were able to stay an average time of 98 minutes after this preliminary treatment. In view of the variability previously mentioned, further study of this method of prevention of decompression pain is needed. Behnke (4) has observed that five hours preliminary denitrogenation by breathing oxygen (without exercise) was sufficient to enable his subjects to stay at 37,000 feet for six hours and at 40,000 feet for two hours without immediate pain or delayed fatigue reactions. It is still an open question whether denitrogenation affords absolute protection against decompression pain at 35,000 to 40,000 feet equivalent altitude. The answer is of great interest in regard to the etiology of decompression sickness.

To understand the above results we require more direct evidence of the mechanism by which gas bubbles produce joint pain and other

symptoms. In Evelyn's study (2) of medical students X-ray photographs were made of joints, skull, and chest to detect large gas bubbles. His results emphasize the constancy of the occurrence of gas bubbles in the joints of all subjects decompressed to an equivalent altitude of 20,000 feet or above. In contrast, the subjective symptoms such as joint pain were variable in occurrence, were not prevalent below 30,000 feet, and had no detectable relation to the presence or absence of large air bubbles. A joint capsule can be distended by gas without any pain and Evelyn states that a joint with undetectable amounts of gas can be painful. He suggested that small bubbles in especially sensitive regions might still be the cause of the pain. These results, though unconfirmed, certainly show that gross mechanical distortion of a joint space by bubbles is not sufficient or necessary to produce pain. A further significant observation was the appearance of large bubbles in the joints at 30,000 feet, even after an hour of denitrogenation by exercise and oxygen breathing. From this evidence, if the gas bubble theory of joint pains is to hold, the symptoms must be attributed to small bubbles in special locations.

Carmichael (3), working at Farnsboro with Matthews, came to the conclusion that peripheral pain symptoms were really due to brain lesions. These lesions were thought to be results of tiny gas bubbles in critical locations in the brain. This hypothesis was also advanced to explain cutaneous phenomena which were thought to be neurotrophic effects due to irritation and distortion of nerve cells by bubbles or to nerve asphyxia produced by intravascular bubbles shutting off the circulation to brain cells. This hypothesis accounts for the occasional convulsions, paralysis or systemic collapse but it seems probable that bubbles in more peripheral locations are also important.

Experiments have been quoted that indicate only limited protection from joint pains by nitrogen elimination. Nevertheless, the idea persists that nitrogen is the most important agent in these incapacitating effects of decompression. Thus, it has been suggested that the rapidity with which the body eliminates its nitrogen is an inverse measure of susceptibility to symptoms due to decompression (8,9,10). Using radio-active argon, the rate of uptake and elimination of an inert gas by tissues have been measured in men. Theoretically and experimentally, the time course of this gas exchange was described by an exponential function involving one rate constant. Slow rates of elimination were correlated with susceptibility to decompression sickness and a rapid rate indicated an immune subject. This result emphasizes again the importance of nitrogen elimination and thus is apparently in disagreement with the demonstration that denitrogenation cannot entirely prevent painful reactions to decompression. Further examination of this test is in progress.

A more detailed investigation of the rate of nitrogen clearance from various tissues has been made (24). The subjects were patients and were older than the age group of primary interest. The results, however, are certainly indicative of the relative rates of nitrogen clearance in young healthy men. When the subjects breathed pure oxygen the nitrogen content of the arterial blood fell to about 10 - 20% of its initial value in two to three minutes.

This rate is probably determined by the rate of elimination of nitrogen from the alveolar air. At first, the arterial venous difference in nitrogen content is large. This is interpreted as the period of rapid loading of the N2 into the blood in the capillaries. In about ten to fifteen minutes the nitrogen contents of the arterial blood and, for example, the internal jugular veins are almost equal. At this time, the limiting factor in N2 clearance seems to be the diffusion of N2 from tissues to the circulating blood. Thus, the N2 content of vascular fluids rapidly decreases but various tissue fluids may still have a high nitrogen content. Synovial fluid from the knee joint or cerebrospinal fluid are cleared of nitrogen much more slowly. Thus, cerebrospinal fluid nitrogen is decreased 50% after one hour of breathing pure oxygen. This observation confirms the earlier findings reported from England (30).

The slow elimination of this substance from joint spaces is probably related to the prevalence of symptoms at such locations and the lack of complete protection from pain by initial breathing of oxygen for short times. The rate of elimination of nitrogen from the hand and arm is speeded by warming and slowed by cooling. This is further evidence for a relation between painful symptoms and nitrogen content of tissues because the incidence of incapacitating symptoms is increased at - 10°F. as compared to 70°F. (22).

These studies emphasize the inequality of rates of nitrogen elimination from various tissues. In this respect the results do not support the view that inert gas elimination can be measured by one rate constant (8,9). However, both of these researches (8,24) suggest that rate of inert gas elimination is an important factor in determining susceptibility of men to decompression sickness. Whether or not this factor is sufficiently dominant to be used as a measure of susceptibility remains to be determined. There are at present at least two facts which do not fit in with this idea. First, if rate of N2 elimination were a major factor, then the rate of climb in a decompression test should have a marked influence on incidence of symptoms. Two research groups have reported that rates of ascent up to 4,000 feet per minute do not modify the results (2,21). In the second place the susceptibility of an individual to incapacitating degrees of decompression sickness is extremely variable. No such variability in the individuals gas elimination rate has been reported. It is, however, possible that such daily variability in elimination of inert gas does occur at, for example, 35,000 feet and not at sea level. From Evelyn's X-ray studies it is known that gas bubbles always form above 20,000 feet. Once bubbles have formed, the conditions for inert gas elimination are changed because the gas must now re-dissolve before it can escape to the blood. In addition, the bubbles may be expected to produce varying degrees of circulatory arrest, thus interfering with elimination of the dissolved inert gas. The first factor could be an extremely variable influence from day to day because of differences in size and number of bubbles. The second effect likewise would seem to be of an inconstant nature. Perhaps further experiments will show whether or not such a mechanism accounts for the variable susceptibility of the individual.

All methods for prevention of decompression pain are centered around elimination of nitrogen from the body. They have not been entirely

practical or successful. At present, personnel for high altitude flying are, therefore, selected according to their ability to withstand decompression to an equivalent altitude of 35,000 feet for four hours (7). A completely satisfactory routine for this selection is not available because of the variable response of the individual from day to day. Further research into the origin of these incapacitating symptoms of decompression sickness and their variable occurrenc is urgently needed. To make the most susceptible subject as immune as the least susceptible of his fellows; or even to make him always as immune as he himself is on some days would be an immense practical advantage for future high altitude missions.

STUDIES ON ANIMALS

The reactions of man to decompression define a major problem in aviation medicine. The object of corresponding experiments on animals is to analyze the mechanism by which these reactions are produced and thus to develop a rational basis for control and alleviation of incapacitating symptoms.

Unfortunately, most of the work on small animals has been done under conditions that are not comparable to those that produce the characteristic symptoms of decompression sickness in man. Thus, in most animal experiments acute reactions to rapid decompression have been studied because small animals do not react markedly to slow rates of decompression. Therefore, it may well be that the results of these studies have little bearing upon the previously defined problem of decompression sickness in man. For, in experiments on men the rate of decompression, up to 4,000 feet per minute, seems to be of minor importance in producing the characteristic reactions.

The principal result of studies on animals is the demonstration that gas bubbles do occur in intravascular and intercellular regions of the body after rapid decompression. However, no experimental demonstration has been forthcoming of the mechanism by which gas bubbles produce the reactions to decompression. It is sometimes assumed that the bubbles cause cellular anoxia and death by blocking the circulation to critical regions of the brain. Furthermore, the prevalence of pulmonary congestion and oedema in explosively decompressed animals led to the idea that death may sometimes be caused by lung damage. However, despite extensive post mortem studies of animals killed by decompression, the cause of their death remains uncertain (11, 12). The principal pathological findings are (12):

1. Lungs
   - (a) Vascular Congestion
   - (b) Oedema
   - (c) Distended peri-arterial lymhatics
   - (d) Occasional rupture of alveolar walls

2. Watery vacuoles in liver.

3. Congestion of cerebral surface veins.

4. No histological damage to brain.

5. Gas bubbles in

   (a) Cerebrospinal fluid
   (b) Tendon sheats
   (c) Cardiovascular system

The customary endpoint for testing an animal's reaction to decompression is to adjust the equivalent altitude and rate of decompression so that some of the animals are killed. Thus, the efficiency of various preventive measures can be estimated in terms of reduced mortality. Thus far, preliminary denitrogenation has produced a significant decrease in death rate of explosively decompressed animals (13), but there is no way to judge whether this protection is comparable in degree with that afforded human subjects by similar procedures. Furthermore, death has occurred in animals denitrogenated by breathing oxygen three hours (17).

One result of this type of research has been to relate the susceptibility of different species of animals to their physical size. The relative immunity of the smaller animals is in part explained by their brief circulation time and small volume (14). Thus, it is thought that the body fluids of small animals cannot become supersaturated with gas except by extremely rapid decompression (to 45,000 feet in 0.4 - 1.0 seconds). Therefore, to produce marked reactions such as convulsions or death in small animals, the rate of ascent to altitude seems more important than the total duration of stay at altitude. These relations are opposite to those reported for humans when incapacitating pain in employed as an index of response to decompression. Henco, the extreme procedure frequently used in animal experiments may not yield an explanation for the kind and degree of reaction that is of principal interest in studies of man.

A promising series of animal experiments has been undertaken by I. deBurgh Daly and his co-workers at the University of Edinburgh (15). They have observed that noticeable reactions are produced in some animals by degrees of decompression that do not kill. By subjecting a series of animals to decompression, they propose to separate them into two groups on the basis of the presence or absence of reactions. These two groups of animals will then be subjected to a variety of physiological and biochemical tests to determine the basis for their difference in susceptibility to reactions of decompression. The proposed procedure, if successful, will be a direct attack on the important problem of differential susceptibility in animals of the same species. The immediate objective of these experiments is to develop tests for susceptibility to decompression sickness that are based upon the fundamental causes.

Bibliography

1. Advances in Aviation Physiology
Aero Medical Research Laboratory, Wright Field
Exp. -M-54-653-74 Dec. 2, 1941

2. The Effects of Low Barometric Pressure on the Health of the Adult Male in the Age Group 19-32
Flight Lt. Kenneth Evelyn, University of Western Ontario Medical School, London, Ontario
Report No. 19, July 3, 1941
National Research Council, Division of Medical Sciences, Committee on Aviation Medicine.

3. Chronological Development of Symptoms in "Bends" in Subjects Breathing Pure Oxygen, with a Neurological Report
B. H. C. Matthews and C. A. Carmichael
C. A. M. Report No. 22 Oct. 5, 1940

4. Investigations Concerned with Problems of High Altitude Flying and Deep Diving; Application of Certain Findings Pertaining to Physical Fitness to the General Military Service.
Lieutenant Commander Albert R. Behnke, Jr., Medical Corps, U. S. Navy
The Military Surgeon, vol. 90, page 9, January 1942

5. Effects of Denitrogenation on Aeroembolism
Aero Medical Research Laboratory, Wright Field
Exp.-M-54-653-44C Jan. 12, 1942

6. Occurrence of Aeroembolism
Aero Medical Research Laboratory, Wright Field
Exp.-M-54-653-44 July 1, 1941

7. Incidence of Symptoms and Duration of Stay at Various High Altitudes
School of Aviation Medicine, Randolph Field

8. The Uptake of Inspired Radioactive Argon. A Method of Determining the Efficiency of Inert Gas Exchange in Man.
Hardin Jones, Robert Smith, Norman Sears, Chien Wu, John Larkin, Robert French, Joseph Hamilton, and John Lawrence.
C. A. M. No. 51 May 29, 1942

9. Theoretical Considerations of Gas Exchange.
Chien-Shiung Wu
C. A. M. Report No. 52 May 29, 1942

10. Aeroemphysema - Animal Investigation
Hardin Jones, Robert Smith, Norman Sears, Chien Wu, John Larkin, Robert French, Joseph Hamilton, and John Lawrence.
C. A. M. Report No. 53, May 29, 1942

11. Histological Changes in the Lungs of Rabbits Decompressed to 40,000 - 47,000 Feet
F. P. R. C. No. 317 by O. A. Trowell (M.D.)

12. See F. P. R. C. Reports Nos. 316, 317, 238, 345, 347, 349, or summary in F. P. R. C. No. 421

13. Observations on Gas Bubble Formation and Lung Damage in Animals Rapidly Decompressed to 43,000 - 47,000 Feet
F. P. R. C. No. 316 Catherine O. Hebb

14. An Investigation into the Cause of "Bend" and Other Ill Effects in Animals Subjected to Pressures Below Atmosphere
F. P. R. C. No. 100 I. deBurgh Daly, P. Eggleton, S. R. Elsdon

15. Proposed Researches by Professor I. deBurgh Daly
F. P. R. C. Report No. 343

16. Observations on Guinea Pigs Explosively Decompressed to Equivalent Heights of 41,000 to 45,000 Feet
J. Fegler
F. P. R. C. Report No. 349

17. Interim Report of Work Carried Out for Physiology Department, Edinburgh University
F. P. R. C. No. 299 by C. O. Hebb

18. Incidence of Decompression Illness in the Classification of Aviation Personnel
Oxygen Indoctrination Unit, School of Aviation Medicine, Medical Department, Naval Air Station Pensacola, Florida
C. A. M. Report No. 65 Aug. 18, 1942

19. Symptoms Experienced During Repeated Low Pressure Chamber Flights and their Significance for High Altitude Classification
School of Aviation Medicine, Randolph Field, Texas
Report No. 57 July 1, 1942 (Also C.A.M. Report No. 71)

20. Repeated Low Pressure Chamber Flights as an Improved Procedure for High Altitude Classification
School of Aviation Medicine, Randolph Field, Texas
Report No. 67 July 2, 1942 (Also C.A.M. Report No. 69)

21. Certain Advantages of a simulated Flight at 38,000 Feet For High Altitude Classification
School of Aviation Medicine, Randolph Field, Texas
Report No. 1, Project 14 Sept. 4, 1942

22. The Effect of Low Temperature on Decompression Sickness
Fraser & Rose
Min. Exec. Assoc., Comm. Aviation Med. Research N.R.C.C.
Feb. 14, 1942, Appendix I

23. The Time Distribution of Symptoms at 35,000 and 38,000 Feet in the Low Pressure Chamber
School of Aviation Medicine, Randolph Field, Texas
Report No. 1, Project No. 71 Sept. 5, 1942

24. Nitrogen Exchange in Tissue Components of Man
E. B. Ferris, W. E. Mollo, and H. W. Ryder
Department of Internal Medicine, University of Cincinnati College of Medicine
C. A. M. Report No. 60 July 15, 1942

25. Minutes of the Sixth Meeting of the F. P. R. C.
October 4, 1939
F. P. R. C. Report No. 61

26. Minutes of the Seventh Meeting of the F. P. R. C.
December 20, 1939
F. P. R. C. Report No. 81

27. Minutes of the Tenth Meeting of the F.P.R.C. May 15, 1940
F. P. R. C. Report No. 145

28. Minutes of the Eighteenth Meeting of the F.P.R.C.
June 4, 1941
F. P. R. C. Report No. 309

29. Minutes of the Nineteenth Meeting of the F.P.R.C.
July 16, 1941
F. P. R. C. Report No. 327

30. Minutes of the Seventeenth Meeting of the F.P.R.C.
May 7, 1941
F. P. R. C. Report No. 298

31. Minutes of the Eleventh Meeting of the F.P.R.C.
June 28, 1940
F. P. R. C. Report No. 163