



# Specialist Medical Review Council

## Reasons for Decision

Section 196W  
Veterans' Entitlements Act 1986

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**Re: Statements of Principles Nos. 31 and 32 of 2001  
Concerning Hypertension**  
Matter No.2001/2  
Requests for review declaration No 6

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### DECISION

1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 31 of 2001 made under subsection 196B (2) of the *Veterans' Entitlements Act 1986* (the VEA), and the RMA Statement of Principles No. 32 of 2001 made under subsection 196B (3) of the VEA, both in respect of hypertension, the Specialist Medical Review Council (the Council) declares, under subsection 196W (4) of the VEA, that it is of the view that there is sound medical-scientific evidence on which the RMA could have relied to amend the Statements of Principles in force in respect of hypertension, particularly as regards occupational or work related stress consequent upon working in a high demand, low decision latitude or control job. The Council, pursuant to section 196W(4)(d) of the VEA, remits the matter to the RMA for reconsideration and:

- (a) directs that the RMA reconsider the three biological markers of stress in hypertension, being the interrelationship with the sympathetic nervous system, the brain transmitter studies, and the adrenaline hypothesis, all of which are more particularly described in paragraph 49 below; and
- (b) recommends that the RMA have regard to:
  - (i) the list of key references referred to by Professor Esler, set out in paragraphs 54 and 55 below;
  - (ii) the article by Drs Schnall, Pickering and Schwartz referred to by the applicant, set out in paragraph 56 below;

- (iii) any further information which has become available to the RMA since the determination of Statements of Principles Nos. 31 and 32 of 2001; and
- (iv) any further information which may become available to the RMA between the date of the Council's Declaration and the completion by the RMA of its reconsideration.

## **FINDINGS ON MATERIAL QUESTIONS OF FACT**

### **Background of events giving rise to the review**

2. On 24 May 2001, the RMA under subsections 196B(2) and (3) of the VEA determined the Statements of Principles being instruments numbered 31 and 32 of 2001 concerning hypertension.
3. On 4 June 2001 and 18 June 2001, in accordance with section 196D of the VEA and sections 46A and 48 of the *Acts Interpretation Act 1901* the two Statements of Principles were tabled in both the House of Representatives and in the Senate.
4. On 6 June 2001 the making of those instruments was notified in the Gazette (No. 22, p.1545).
5. On 13 June 2001 the Department of Veterans' Affairs received an application for review of Statements of Principles Nos. 31 and 32 of 2001 (the application). Specifically, the application was concerned with a decision of the RMA of 24 May 2001 refusing to add 'Occupational or Work Related Stress' as a factor to Statement of Principles Nos. 31 and 32 of 2001 concerning hypertension.
6. The application was accompanied by a detailed submission from the applicant and reports of Dr Gordon and Dr Stevenson. The applicant was critical of the failure of the RMA to include 'Occupational or Work Related Stress' as one of the criteria within the Statements of Principles concerning hypertension. The applicant based this objection on what he considered to be 'the failure of the RMA to consider the relationship of the latest sound medical-scientific evidence in providing a 'direct link to stress and hypertension''.
7. The applicant particularly but not exclusively relied upon the article co-authored by Professor Murray Esler: (Rumantir et al) *'The 'adrenaline hypothesis' of hypertension revisited: evidence for adrenaline release from the heart of patients with essential hypertension'* Journal of Hypertension 2000, Vol 18 No 6, p717-723.
8. In quoting this reference the applicant drew attention to the fact that the paper had been before the RMA at the time of its promulgation of Statements of Principles Nos. 31 and 32. It was his contention that:
  - (a) due weight had not been accorded to this reference; and

- (b) if due weight had been given to this reference, ‘work related stress’ would have been able to be listed in the relevant Statements of Principles as a factor that as a minimum must exist.’

9. The Repatriation Commission also made a submission to the Council.

10. The Council convened for the purpose of this review on 28 November 2001.

### **The Specialist Medical Review Council**

11. The Council is a body corporate established under section 196V of the VEA, and consists of such number of members as the Minister for Veterans' Affairs determines from time to time to be necessary for the proper exercise of the function of the Council as set out in the VEA. The Minister must appoint one of the Councillors to be the Convenor. When a review is undertaken of a Statement of Principles made by the RMA, the Council is constituted by 3 to 5 Councillors selected by the Convenor. When appointing Councillors, the Minister is required to have regard to the branches of medical science expertise which would be necessary for deciding matters referred to the Council for review.

12. Professor Alex Cohen AO, MD, FRACP was the Convenor of the Council for this review. The other members of the Council were: Professor Ian Puddey, Professor of Medicine, Head of the Department of Internal Medicine at Royal Perth Hospital, University of Western Australia; Emeritus Professor Scott Henderson, visiting Senior Specialist, Psychiatry Unit The Canberra Hospital; Professor Murray Esler, Head of Cardiovascular Neuroscience Laboratory at the Baker Institute, Associate Director of the Alfred and Baker Medical Institute Unit, Alfred Hospital, and Professor of Medicine Monash University Melbourne; and Dr Jonathan Phillips, Consultant Psychiatrist, and Chairman of the Committee of Presidents of the Australian Medical Colleges.

### **The Legislation**

13. The legislative scheme for the making of Statements of Principles is set out in Part X1A and X1B of the VEA.

14. The functions and powers of the Council must be seen in light of the function and purpose of Statements of Principles in the scheme of the VEA. The significance of Statements of Principles to claims under the VEA for pensions in relation to eligible service is apparent from sections 120A and 120B of the VEA. Section 120 is also of importance.

### **Sound Medical-Scientific Evidence**

15. Fundamental to Statements of Principles is the concept of ‘sound medical- scientific evidence’ which has been defined in section 5AB of the VEA. Information about a particular kind of injury, disease or death is taken to be sound medical-scientific evidence if:

- (a) the information

- (i) is consistent with material relating to medical science that has been published in a medical or scientific publication and has been, in the

opinion of the Repatriation Medical Authority, subjected to a peer review process;

or

- (ii) in accordance with generally accepted medical practice, would serve as the basis for diagnosis or management of a medical condition; and
- (b) in the case of information about how that injury, disease or death may be caused - meets the applicable criteria for assessing causation currently applied in the field of epidemiology.

16. Under section 196W of the VEA, the Council can only reach the view that a Statement of Principles should be amended on the basis of sound medical-scientific evidence.

### **Oral Submissions**

#### **Applicant's submissions**

17. The applicant represented himself.

18. The initial part of his presentation comprised a detailed account of his own personal and family history prior to, during, and subsequent to his enlistment.

19. In support of his submissions that 'Occupational and Work place Stress' should be included in the Statements of Principles concerning hypertension, the applicant relied upon the following key passages. (The Council notes that the passages set out below are as put to the Council by the applicant. In some cases the passages are not directly quoted from the references cited, and in others, it is not clear that words from the references cited have been omitted. Rather, in some instances, the passages are a combination of reference material, and the applicant's own comment). All papers relied upon by the applicant are referenced below at paragraph 63.

- From numerous clinical studies a phenotype of neurogenic human hypertension has been delineated, and testing of the heritability of this neurogenic variant of essential hypertension has commenced. The sympathetic nervous variation is manifest in an increase in muscle sympathetic nerve firing and high rates of spill-over of noradrenaline from the kidneys and heart. Coupled with the resultant high plasma resin activity, an accentuated haemodynamic response to pharmacological adrenaline blockade, a high heart rate, and in some cases an elevated cardiac output. (at page 645) Professor Murray Esler '*Sympathetic Activity in Experimental and Human Hypertension*' Handbook of Hypertension 1997 Vol 17: Pathophysiology of Hypertension, p 629-673.
- Although the concept that in some patients essential hypertension may arise by psychosomatic mechanisms is not entirely unproven there is a wealth of supporting experimental and clinical evidence. (*ibid* at page 647).

- Evidence for adrenaline release from the heart of patients with essential hypertension provides sound, medical-scientific evidence that stress leads to hypertension. The Australian Doctor ... outlines that study and states 'ongoing stress leads to an accumulation of adrenaline which subsequently causes vasoconstriction and hypertension'.
- These include exposure to stressful life events, characteristics of the work environment and social resources. In addition, long term occupational stress, such as the strain of a demanding job that affords little opportunity for decision making, increases risk for CHD, hypertension and stroke. '*National Heart, Lung and Blood Pressure Institute Report of the Task Force on Behavioural Research in Cardiovascular Lung and Blood Health Disease*' 1998 – *Other Social Stresses and Health* at page 8.
- In humans increased prevalence of hypertension is associated with an individual's long term exposure to daily stresses, such as or having high demand and low control jobs. '*National Heart, Lung and Blood Pressure Institute Report of the Task Force on Behavioural Research in Cardiovascular Lung and Blood Health Disease*' 1998– *Sustained Hypertension* at page 64.
- Blood pressure reactions to the uncontrollable task were greater in high than low job strain groups. Job strain is associated with a heightened blood pressure response to uncontrollable but not controllable tasks. (Abstract under Results and Conclusions at page 193) Steptoe *et al* report '*Job Strain, Blood Pressure and Response to Uncontrollable Stress*' The Journal of Hypertension 1999, Vol 17, No 2 p 193-200.
- Several studies have suggested that chronic social conflict is associated with higher blood pressure and ambulatory monitoring has shown that most people have their highest pressure during working hours. Occupational stress can be evaluated as job strain which is a combination of high demands at work with low decision latitude or control. (Abstract at page 9) Dr T Pickering report '*The Effects of the Environmental and Lifestyle Factors on Blood Pressure and the Intermediary Role of the Sympathetic Nervous System*' The Journal of Human Hypertension 1997, 11, Suppl 1 p 9-18.
- Stress can cause hypertension through repeated blood pressure elevations ... Factors affecting blood pressure through stress include white coat hypertension, job strain, social environment and emotional distress ... Although stress may not directly cause hypertension, it can lead to repeated blood pressure elevations which eventually may lead to hypertension. (Abstract at page 38) Kulkarni *et al* '*Stress and Hypertension*' Wisconsin Medical Journal 1998 p 34-38.

- One potentially important factor is work load or the objective level of work demand. Perceptions of work rather than objective work load may be equally important ... This paper discusses Karasek's demand control model of work stress. This model postulates that high levels of job strain arise when high physiological demands at work are coupled with low decision latitude or control over how the job is done. (at page 687) Steptoe '*Ambulatory Blood Pressure and Work*' Handbook of Hypertension Vol. 17: Pathophysiology of Hypertension, 1997, p674-708.
- Moreover, a substantial case control study of working men aged 30 to 60 years, has shown that exposure to job strain substantially increases the likelihood of hypertension ... Work can be a source of chronic stress, with elements such as time pressure, heavy responsibility, conflicting social relationships and job insecurity having potent effects. *Steptoe ibid* at pages 688 and 692 respectively.

20. The applicant then introduced reports from Dr Richard Gordon referable to the applicant's own circumstances. Notwithstanding the applicant was allowed to present this material to the Council, it should be noted that the Council does not examine and review the application of individuals on the basis of personal history of illness.

21. In reply to the Repatriation Commission's submissions, the applicant submitted that the material upon which he relied (as referred to above) met the criteria laid down by Bradford Hill.

### **Repatriation Commission's Submissions**

22. Dr John Kelley, a Medical Officer with the Department of Veterans' Affairs, represented the Repatriation Commission. He was accompanied by Dr Keith Horsely. Dr Kelley was the principal author of the Repatriation Commission's written submission to the Council.

23. The Repatriation Commission drew the Council's attention to the fact that the RMA has carried out three separate formal investigations into stress as a potential cause of hypertension. One of those investigations had incorporated a Consensus Conference, with participation by national and international experts in the field.

24. The Commission noted the RMA had accepted as relevant outcomes of severe stress the conditions of ischaemic heart disease; cerebrovascular accident; anxiety disorder and irritable bowel syndrome. The nature of the stress in these clinical conditions has been specified by the RMA to range from severe stressor; severe psychosocial stressor to specified psychiatric condition. The time interval between stress and onset varies between immediately; within 48 hours; within two years and in the last instance, six months (Repatriation Commission submission paragraph 11).

25. The RMA has not to date included either chronic (situational) stress, such as from job strain, or personality traits as causal factors in any Statements of Principles.
26. Dr Kelley referred to the epidemiological information currently available in the literature as to any causal connection between stress and hypertension. Dr Kelley examined that material in the light of the Bradford Hill criteria with some variations.
27. Dr Kelley submitted there were methodological difficulties in the studies supporting an asserted causation between occupational or workplace stress and hypertension. He submitted that foremost among these was: 'the amorphous and ubiquitous nature of stress and the problems of adequately characterising and measuring stress' (Repatriation Commission Submission, paragraph 17). After outlining a number of primary research studies he conceded: 'Some of these studies provide support for a role of stress in the causation of hypertension but many do not' (Repatriation Commission Submission, paragraph 19).
28. The principal foundation on which Dr Kelley based his submission was the paper ('*Stress, Personality Interactions and Hypertension*', p 133-150 of the proceedings monograph) prepared by Professor Don Byrne in 1998 for the Consensus Conference on Stress and Challenge, Health and Disease. This paper was before the RMA at the time of creation of the current Statements of Principles.
29. Dr Kelley relied upon the opinion of Professor Byrne that there was no sufficient causal connection between stress and hypertension, contending particularly that the association was lacking in strength, consistency and specificity; that appropriate temporality had really not been shown; and that no dose response effect had been adequately demonstrated.
30. Dr Kelley conceded that the review by Andrew Steptoe, '*Ambulatory Blood Pressure and Work*' Handbook of Hypertension Vol. 17: Pathophysiology of Hypertension, 1997, pp 674-708, provided a comprehensive summary of the principal approaches to the investigation of stress and hypertension, and the strongest support for a role for psychosocial factors in the pathogenesis of hypertension. Dr Kelley nevertheless submitted that neither this review, nor any of the others cited, demonstrated workplace stress as an independent risk factor for the development of hypertension.
31. In quoting a number of recent studies – both prospective and cross sectional - Dr Kelley submitted '...that there is material that offers some support for the stress-hypertension hypothesis countered by material that does not support that association' (Repatriation Commission Submission, paragraph 27). In noting the criteria considered by the RMA to be necessary to give credibility to a causal association, Dr Kelley tabled four recent studies that in his submission met these criteria. In none of these was an unequivocal, unadulterated causal connection shown, the studies showing a negative, inverse or mixed outcome.
32. Dr Kelley submitted that: 'Overall the Commission considers that these better quality epidemiological studies offer limited and inconsistent support for a causal association between stress and hypertension, with more negative than positive findings' (Repatriation Commission Submission, paragraph 33).

33. With regard to the ‘adrenaline hypothesis’, Dr Kelley addressed the particular paper cited in the application and agreed that there was support for the notion that ‘stress mediates hypertension via stimulation of the sympathetic nervous system.’ However, he submitted that the paper did not prove that ‘work-related stress’ was responsible for this adrenaline release, nor that this adrenaline release results in sustained hypertension (Repatriation Commission submission paragraph 35).

34. Dr Kelley concluded by submitting that there was a strong body of dissent among the scientific community to the linking of stress and hypertension, based primarily upon the inconclusive findings of some of (what he submitted were) the better quality prospective studies. He stressed the need for further evaluations despite the immense amount of material already in existence.

35. Dr Kelley conceded that there was quite a deal of supportive evidence connecting stress with hypertension. However, he submitted that the whole field is beset by methodological difficulties, and that further prospective studies of job strain were required. For these reasons, taking into account those studies currently available, he reiterated his submission that the association lacks strength, consistency and specificity; that appropriate temporality had been rarely shown; and adequate dose – response effect had rarely been adequately demonstrated. These epidemiological criteria are those which have been ordinarily taken into account in assessing whether there is sufficient sound-medical scientific evidence upon which the RMA may rely when formulating a Statement of Principles.

36. In conclusion Dr Kelley submitted that the current level of information, although suggestive, is not of itself sufficient to support a conclusion that there is sound medical-scientific evidence for inclusion into the Statements of Principles concerning hypertension a factor of ‘occupational or work related stress’. He submitted that further prospective studies of job strain were required.

37. Finally Dr Kelley submitted that (in the event the Council formed the view that amendment to the Statement of Principles for hypertension may be warranted) the matter be remitted to the RMA for its further consideration.

## **REASONS FOR THE COUNCIL’S DECISION**

38. The Council in conducting this review of Statements of Principles Nos. 31 and 32 of 2001 was mindful of its obligation to comply with the decision of the majority in the New South Wales Court of Appeal in *Repatriation Commission v Vietnam Veterans’ Association of Australia NSW Branch Inc. & Ors* (2000) 171 ALR 523 (RC v VVAA) as to the manner in which it should carry out its statutory function. The Council considered the materials before it, for the purpose of determining what constituted the pool of information, being that information which epidemiologists would consider appropriate to take into account. The Council decided that there was no study or other information that was so methodologically flawed that it should, for that or any other reason, be excluded from the pool of information.



Accordingly, all the material that was before the RMA was taken into account by the Council.

39. The Council, after reviewing all the information that was before the RMA, decided to consider only the issue raised by the applicant, i.e. whether ‘occupational or work related stress’ should be included as a factor in the Statements of Principles concerning hypertension.

40. The Council does not believe that its consideration of the material in the pool of information (in order to establish whether there was sufficient sound medical-scientific evidence to include ‘occupational and work related stress consequent upon working in a high demand, low decision latitude or control job’ as a factor in the Statements of Principles concerning hypertension) requires the slavish adoption of a particular set of criteria such as those enunciated by Sir Austin Bradford Hill. Nevertheless, these remain of considerable value.

41. The Council has been at pains to ensure that in considering whether either of the Statements of Principles should be amended, it remained focussed on its task. This was to consider whether the material in the pool of information provided sufficient sound medical-scientific evidence of a causal relationship between ‘occupational or work related stress consequent upon working in a high demand, low decision latitude or control job’ and hypertension to warrant amendment to the Statement of Principles. In this regard the Council was concerned to ensure that the focus remained on evidence of actual causal connections and not simply on trends and possibilities.

42. In this context, it was necessary to clearly define those aspects of the studies that could be regarded as confounding. With respect to hypertension this was very significant, since most studies omitted significant features such as family history, which could well have influenced the outcome. This is further exacerbated in a consideration of stress, given there is no generally agreed upon definition.

43. The Council, after directing itself in accordance with the principles laid down by the New South Wales Court of Appeal in *RC v VVAA*, decided first that there was no material which should be excluded from the pool of information, and that all material which was before the RMA was properly the subject of consideration by the Council. After considering the material in the pool of information, the Council unanimously considered that there was sufficient sound-medical scientific evidence to warrant reconsideration being given to whether there is a causal relationship between the release of adrenaline and hypertension, both acute and chronic, and occupational stress and hypertension.

44. The Council particularly considered the material in support of the adrenaline hypothesis in the causation of sustained hypertension. Notwithstanding this hypothesis was the causative lynch-pin relied upon by the applicant to causally connect stress and hypertension, the Council considers this hypothesis is only one of several ‘links in the chain’, and only one factor in the genesis of hypertension.

45. In this regard, and as noted in paragraph 19 above, the Council took into account that some of the passages, cited by the applicant in support of his submission, did not necessarily accurately reflect the paraphrased text upon which he relied. The most striking and significant example of this was the passage cited in the third dot point of paragraph 19 above.

46. The Council understands that the passage cited was paraphrasing by the applicant of an article by Heather Ferguson in the Australian Doctor, 18 February 2000, p12. The actual passage reads: 'ongoing stress leads to an accumulation of adrenaline which subsequently causes vasoconstriction and hypertension ...results of a study provided evidence that stress led to hypertension'. These are statements attributed by Ms Ferguson to Professor Murray Elser about the paper '*The 'adrenaline hypothesis' of hypertension revisited: evidence for adrenaline release from the heart of patients with essential hypertension*' Journal of Hypertension 2000, Vol 18 No 6, p717-723.

47. Professor Elser's paper (Rumantir *et al*) actually concludes that the adrenaline spillover effect: '...provid[es] provocative but by no means definitive evidence in support of the adrenaline hypothesis of essential hypertension.' It goes on to add a further qualification to the adrenaline hypothesis, highlighted by patients with panic attack disorder who fit well the prerequisites of the hypothesis, but who usually do not have elevated blood pressure.

48. As mentioned above, the Council considers that the adrenaline hypothesis is one of the 'links in the chain' of a potential causal relationship between occupational stress and hypertension. The Council considers there are three biological markers of stress with potential causal links to hypertension, which as a whole constitute sound medical-scientific evidence upon which the RMA could have relied to amend the Statements of Principles in force in respect of hypertension to include as a factor, 'occupational or work related stress consequent upon working in a high demand, low decision latitude or control job'. The Council considers these three markers merit serious reconsideration by the RMA.

49. The three markers are as follows:

- (i) the inter-relationship between stress, of whatever kind, and activation of the sympathetic nervous system, and the corresponding influence on blood pressure. The Council considers that this inter-relationship has been strongly shown. The Council notes there are findings in the medical literature of the presence of chronic activation of the sympathetic nervous system in hypertensives, which is a putative link between stress and hypertension as the mediator;
- (ii) studies of brain transmitters in human hypertension of a type that would be activated in a stress response, based on internal jugular venous sampling in the catheter load. These studies have shown that projections of a particular sort of neurone from the brain stem, noradrenergic neurones from the brain stem to subcortical areas of the forebrain, which are principally the hypothalamus and amygdala, are activated two to threefold above the normal level; and
- (iii) the adrenaline hypothesis, which postulates the idea that if hypertensives have been subjected to continuing high levels of stress,

the adrenaline they release from their adrenal medulla is taken up into sympathetic nerves and released as a co-transmitter or second chemical messenger, and that this, through mechanisms involving release of the main chemical messenger in nerve endings, could initiate hypertension. The finding is that adrenaline is released from both the heart and the kidneys in hypertensives. It is, again, evidence of the exposure of hypertensives to higher ongoing levels of stress.

50. The Council considers the three markers as equally powerful, and in combination, sufficient sound medical-scientific evidence to justify the RMA amending the Statements of Principles to include as a factor 'occupational or work related stress consequent upon working in a high demand, low decision latitude or control job'. The Council notes, however, with respect to the adrenaline hypothesis that the evidence that the adrenaline has actually caused the hypertension, rather than other components of the stress response, remains inconclusive. The Council does not consider that the adrenaline hypothesis alone would justify an amendment to the Statement of Principles. Rather, it considers that the totality of the sound medical-scientific evidence about the three markers is sufficient to justify such an amendment.

51. The Council was satisfied on the basis of the materials before it, and the submissions addressed to it, that there was sufficient evidence of sufficient weight before it upon which the RMA could have relied to amend the Statements of Principles in force concerning hypertension to include 'Occupational or work related stress consequent upon working in a high demand, low decision latitude or control job'. Accordingly, the Council was of the view that the matter should be remitted to the RMA for reconsideration.

52. In remitting a matter to the RMA, it is open to the Council to make directions or recommendations.

53. The Council directs that the RMA reconsider the three biological markers of stress in hypertension, being the interrelationship with the sympathetic nervous system, the brain transmitter studies, and the adrenaline hypothesis, all of which are more particularly described in paragraph 49 above.

54. The Council notes it is possible that some references, considered by Professor Esler to be important, were not drawn to the RMA's attention. Professor Esler, in commenting on the paper prepared by Professor Byrne for the Consensus Conference (upon which Dr Kelley heavily relied) drew attention to the fact that he (Professor Esler) had been a participant at that Conference at which he had presented a paper published as a chapter. Professor Esler considered he had included in this chapter historic and key references that were not included in Professor Byrne's list of references, in support of a causal connection between stress and hypertension. The papers referred to by Professor Esler are listed at paragraph 61.

55. Further, Professor Esler referred to articles (also cited at paragraph 61) on brain transmitter mechanisms, published between 1992 and 1994, which were not included in the RMA's list of the publications it had considered. The Council recommends all these publications listed at paragraph 61 should be considered by the RMA when it conducts its reconsideration.

56. Finally, the applicant referred to a paper by Drs Schnall, Pickering and Schwartz, '*Job Stress as a Factor in Developing Hypertension*', which was not before the RMA. The applicant then submitted to the Review Council copies of four papers by these authors (listed at paragraph 58). The Council recommends the RMA take these papers into account in its reconsideration.

57. The Council recommends the RMA consider these additional studies when reconsidering the three biological markers of a causal connection between 'occupational or work related stress consequent upon working in a high demand, low decision latitude or control job' and hypertension, together with any further information which has become available to the RMA since it determined the Statements of Principles concerning hypertension, and any further information which may become available to the RMA between the date of the Council's Declaration and the completion by the RMA of its reconsideration.

58. The Council is unable to characterise the particular dosage and temporal relationships which may ultimately be specified should the RMA add as a factor in the Statement of Principles concerning hypertension 'occupational or work related stress consequent upon working in a high demand, low decision latitude or control job.' The Council anticipates that the RMA will consider the dosage and temporal relationships in a similar way to that referred to in paragraph 24 above.

## **DECLARATION**

59. The Council was of the view and declares that there was sufficient sound medical-scientific evidence available to the RMA to justify an amendment of both Statement of Principles No. 31 and Statement of Principles No. 32 of 2001 to include as a factor 'occupational or work related stress consequent upon working in a high demand, low decision latitude or control job.' The Council remits the matter to the RMA for reconsideration in accordance with the direction in paragraph 53 above, and the recommendations set out in paragraphs 54 – 57 above.

## EVIDENCE BEFORE THE COUNCIL

### Documents

60. The material sent to the Council by the RMA (and considered by the Council) was as listed in Appendix A.

61. The material referred to by Professor Esler in paragraphs 54 and 55 above is as listed below, in the order the articles appear in the Repatriation Medical Authority Conference 1998 Monograph and as supplied by Professor Esler.

#### *RMA Conference material – paragraph 54 above*

1. Selye H. The Stress of Life. 1956. New York, McGraw-Hill.
2. Goldstein DS. Clinical assessment of sympathetic responses to stress. *Ann NY Acad Sci* 1995;771;570-593.
3. Hagbarth K-E, Vallbo AB. Pulse and respiratory grouping of sympathetic impulses in human muscle nerves. *Acta Physiol Scand* 1968;74:96-108.
4. von Euler US, Hellner S, Purkhold A. Excretion of noradrenaline in the urine in hypertension. *Scand J Clin Lab Invest* 1954;6:54-59.
5. Esler M, Jackman G, Bobik A, Kelleher D, Jennings G, Leonard P, Skews H, Korner P. Determination of norepinephrine apparent release rate and clearance in humans. *Life Sciences* 1979;25:1461-1470.
6. Esler M, Jennings G, Korner P, Blombery P, Sacharias N, Leonard P. Measurement of total and organ-specific norepinephrine kinetics in humans. *Am J Physiol* 1984;247 (Endocrinol Metab 10):E21-E28.
7. Esler M, Jennings G, Lambert G, Meredith I, Horne M, Eisenhofer G. Overflow of catecholamine neurotransmitters to the circulation: Source, fate and functions. *Physiological Reviews* 1990;70:963-985.
8. Esler MD, Thompson JM, Kaye DM, Turner AG, Jennings GL, Cox HS, Lambert GW, Seals DR. Effects of aging on the responsiveness of the human cardiac sympathetic nerves to stressors. *Circulation* 1995;91:351-358.
9. Esler M, Jennings G, Lambert G. Measurement of overall and cardiac norepinephrine release into plasma during cognitive challenge. *Psychoneuroendocrinology* 1989;14:477-481.
10. Lown B, Verrier RL. Neural activity and ventricular fibrillation. *New England Journal of Medicine* 1976;294:1165-1170.

11. Anderson EA, Sinkey CA, Lawton WJ, Mark AL. Elevated sympathetic nerve activity in borderline hypertensive humans: evidence from direct intraneural recordings. *Hypertension* 1989;14:177-183.
12. Esler M, Jennings G, Korner P, Willett I, Dudley F, Hasking G, Anderson W, Lambert G. The assessment of human sympathetic nervous system activity from measurements of norepinephrine turnover. *Hypertension* 1988;11:3-20.
13. Esler M. Hyperadrenergic and "labile" hypertension. In: Swales J ed. *Textbook of Hypertension*. London: Blackwell, 1994:741-749.
14. Moran MJ, Kennedy HL, Padgett NE. Do borderline hypertensive patients have labile blood pressure? *Ann Intern Med* 1981;94:466-468.
15. Mancia G, Ferrari G, Gregorini L, Parati G, Pomidossi G, Bertinieri G, Grassi G, di Rienzo M, Pedotti A, Zanchetti A. Blood pressure and heart rate variabilities in normotensive and hypertensive human beings. *Circ Res* 1983;53:96-104.
16. Perini C, Muller FB, Rauchfleisch U, Battegay R, Hobi V, Buhler FR. Psychosomatic factors in borderline hypertensive subjects and offspring of hypertensive parents. *Hypertension* 1990;16:627-634.
17. Julius S, Jones K, Schork N, Johnson E, Krause L, Nazzaro P, Zemva A. Independence of pressure reactivity from pressure levels in Tecumseh, Michigan. *Hypertension* 1991;17 (suppl III):12-21.
18. Lindqvist M, Kahan T, Melcher A, Hjemdahl P. Cardiovascular and sympatho-adrenal responses to mental stress in primary hypertension. *Clin Sci* 1993;85:401-409.
19. Geisbock F. Cited in Julius S, Esler M, ed. *The Nervous System In Arterial Hypertension*. Springfield, Illinois: Charles C Thomas, 1976; xii.
20. Esler M, Julius S, Zweifler A, Randall O, Harburg E, Gardiner H, DeQuattro V. Mild high-renin essential hypertension: a neurogenic human hypertension ? *New Engl J Med* 1977;296:405-411.
21. Harburg E, Erfurt JC, Hauenstein LS, Chape C, Schull WJ, Schork MA. Socio-ecological stress, suppressed hostility, skin colour, and black-white male blood pressure: Detroit. *Psychosom Med* 1973;35:276-296.
22. Perini C, Muller FB, Rauchfleisch U, Battegay R, Buhler FR. Hyperadrenergic borderline hypertension is characterized by suppressed aggression. *J Cardiovasc Pharmacol* 1986;8 (Suppl 5):53-56.

23. Poulter NR, Khaw KT, Hopwood BEC, Mugambi M, Peart WS, Rose G, Sever PS. The Kenyan Luo migration study: observations on the initiation of the rise in blood pressure. *BMJ* 1990;300:967-972.
24. Timio M, Verdechioa P, Rononi M, Gentili S, Francucci B, Bichisao E. Age and blood pressure changes: a 20 year follow-up study of nuns of a secluded order. *Hypertension* 1988;12:457-461.
25. Henry JP, Grim CE. Psychosocial mechanisms of primary hypertension. *J Hypertens* 1990;8:783-793.
26. Alexander F. Emotional factors in essential hypertension. *Psychosom Med* 1939; 1:173-179.
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