PATHOPHYSIOLOGICAL BASIS OF CEREBRAL ARTERIAL AIR EMBOLISM

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LIST OF CONTENTS

LIST OF FIGURES ix
LIST OF TABLES xii
SUMMARY xv
PUBLICATIONS ARISING FROM THESE STUDIES xvii
DECLARATION xix
ACKNOWLEDGMENTS xxi

CHAPTER 1. BACKGROUND AND REVIEW OF THE LITERATURE 1
1.1. OVERVIEW 1
  1.1.1. Scope of this review 2
  1.1.2. Terminology 2
1.2. HISTORICAL ASPECTS OF GAS EMBOLISM 4
1.3. THE CEREBRAL CIRCULATION - BASIC PHYSIOLOGY 7
  1.3.1. Cerebrovascular anatomy 8
  1.3.2. Cerebrovascular carbon dioxide reactivity 71
  1.3.3. Cerebrovascular oxygen response 12
  1.3.4. Cerebral perfusion pressure 12
  1.3.5. Capillary cycling or capillary recruitment? 13
      1.3.5.1. Capillary cycling 13
      1.3.5.2. Capillary recruitment 13
      1.3.5.3. Embolism and capillary cycling or recruitment 14
  1.3.6. Neural mechanisms of CBF regulation 15
  1.3.7. The blood-brain barrier 15
  1.3.8. Role of the vascular endothelium in the regulation of CBF 16
  1.3.9. Cortical-somatosensory evoked responses 18
1.4. CAUSES OF GAS EMBOLISM 20
  1.4.1. Dysbaric causes of arterial gas embolism 21
      1.4.1.1. Decompression illness 21
      1.4.1.2. Decompression schedules 22
      1.4.1.3. Gas gradients 25
      1.4.1.4. Limb bends 26
      1.4.1.5. Decompression "folklore" 26
      1.4.1.6. Decompression illness and "silent bubbles" 27
      1.4.1.7. Spinal cord decompression sickness 28
      1.4.1.8. Barotrauma 29
      1.4.1.9. Submarine escape training 30
1.4.2. Iatrogenic causes of gas embolism
1.4.3. Traumatic causes of gas embolism
1.5. Classification of gas embolism
1.5.1. Arterial gas embolism
1.5.2. Venous gas embolism
1.5.3. Paradoxical gas embolism
1.5.3.1. The patent foramen ovale
1.5.3.2. The Valsalva manoeuvre
1.5.3.3. Foramen ovale and diving
1.5.3.4. Failure of the pulmonary filter
1.5.3.5. Effects of posture
1.5.3.6. Effects of ventilation
1.5.3.7. Effects of immersion
1.6. Behaviour of gaseous phase cases in the circulation
1.6.1. Bubble passage through cerebral vessels
1.6.2. Coalescence
1.6.3. Stabilisation of bubbles
1.6.4. Effects of air dose
1.7. Effects of gaseous phase cases on the circulation
1.7.1. Air in the circulation
1.7.2. Oxygen bubbles in the circulation
1.7.3. Carbon dioxide bubbles in the circulation
1.7.4. Effects of gas bubbles on the blood constituents
1.7.4.1. Effects of gas bubbles on complement factors
1.7.4.2. Effects of gas bubbles on red blood cells
1.7.4.3. Effects of gas bubbles on platelets
1.7.4.4. Effects of gas bubbles on leukocytes
1.7.4.5. Ischaemia-reperfusion injury and CAGE
1.7.4.6. Disseminated intravascular coagulation
1.7.4.7. Is CAGE a disseminated intravascular coagulopathy?
1.8. Pathophysiological effects of CAGE
1.8.1. Damage to vascular endothelial cells caused by CAGE
1.8.2. Cerebral vessel dilation caused by CAGE
1.8.3. Damage to the blood-brain barrier caused by CAGE
1.8.4. Cerebral oedema caused by CAGE
1.8.5. Effects of CAGE on CBF
1.8.6. Damage to the brain parenchyma caused by CAGE
1.8.7. Relapse after initial improvement from CAGE
1.9. Outcome and treatment after CAGE
1.9.1. Treatment of CAGE
1.9.1.1. Trendelenburg position
1.9.1.2. Hyperbaric therapy
1.9.1.3. Hyperbaric oxygen therapy
1.9.1.4. How does hyperbaric oxygen treatment work?
1.9.1.5. Pharmacological and other treatments
1.10. Experimental methods used to study CAGE
   1.10.1. Studies in humans  79
   1.10.2. Studies in animals  81
1.11. CAGE pathophysiology  82
1.12. Proposed studies  84
   1.12.1. Hypothesis and aims  85

CHAPTER 2. METHODS AND MATERIALS USED  87
2.1. ANIMALS  87
2.2. ANESTHESIA  87
2.3. SURGERY  88
2.4. Cortical somatosensory evoked responses  92
   2.4.1. Method for cortical somatosensory evoked responses  93
   2.4.2. Spreading depression  95
2.5. General blood flow  96
   2.5.1. Microsphere technique  96
   2.5.2. Laser Doppler flowmetry  97
   2.5.3. Tracer accumulation methods  98
   2.5.4. Clearance methods, particularly hydrogen clearance  99
      2.5.4.1. Compartmental analysis  103
      2.5.4.2. Stochastic analysis  104
      2.5.4.3. Initial slope index analysis  104
      2.5.4.4. The virtual ground circuit  104
      2.5.4.5. Application of the hydrogen clearance technique  105
2.6. Pial arteriolar diameter  108
2.7. ANALYSIS OF RESULTS  109
   2.7.1. The Bonferroni method  110
2.8. EXPERIMENTAL PLAN  110
   2.8.1. Sequencing of experiments  111
   2.8.2. Sequence of steps  111

CHAPTER 3. A MODEL OF CAGE  119
3.1. Effects of gas embolism on brain blood flow and function  119
3.2. METHODS  119
3.3. RESULTS  120
   3.3.1. General observations  120
   3.3.2. Right CBF  121
   3.3.3. Left CBF  121
   3.3.4. Pial artery diameter  122
   3.3.5. Pial venous diameter  122
   3.3.6. CSER AP₂  123
   3.3.7. Relationship between CSER AP₂ and CBF  123
CHAPTER 4. INCREASING DOSES OF AIR

4.1. EFFECTS OF INCREASING DOSES OF INTRACAROTID AIR ON CEREBRAL BLOOD FLOW AND BRAIN FUNCTION

4.2. METHODS

4.3. RESULTS

4.3.1. General observations

4.3.2. Right CBF

4.3.3. Left CBF

4.3.4. Pial arterial diameter

4.3.5. Pial venous diameter

4.3.6. CSER AP₂

4.3.7. Relationship between CSER AP₂ and CBF

4.4. DISCUSSION

CHAPTER 5. EFFECTS OF GRANULOCYTE DEPLETION

5.1. AIR EMBOLSM OF THE BRAIN IN RABBITS PRE-TRAINED WITH MECAZOLAMINE

5.2. METHODS

5.3. RESULTS

5.3.1. General observations

5.3.2. Right CBF

5.3.3. Left CBF

5.3.4. Pial arterial diameter

5.3.5. Pial venous diameter

5.3.6. CSER AP₂

5.4. DISCUSSION

CHAPTER 6. MODIFICATION OF LEUKOCYTE ADHESION

6.1. STUDIES WITH DEXTRAN SULPHATE

6.2. MATERIALS AND METHODS

6.3. RESULTS

6.3.1. General observations

6.3.2. Right CBF

6.3.3. Left CBF

6.3.4. Pial arteriolar diameter

6.3.5. Pial venous diameter

6.3.6. CSER AP₂

6.3.7. Relationship between CSER AP₂ and CBF

6.4. DISCUSSION

CHAPTER 7. DISCUSSION AND CONCLUSIONS

7.1. THE CONVENTIONAL VIEW

7.2. PREVIOUS STUDIES

7.2.1. Prevention of CAGE

VI
7.2.2. The patent foramen ovale 183
7.2.3. Hyperbaric oxygen therapy 184
7.2.4. Pharmacological treatment of CAGE 185
7.2.4.1. CAGE and lignocaine 185
7.2.4.2. CAGE and steroids 185
7.2.4.3. CAGE and the 'triple combination' 186
7.2.4.4. CAGE and kaldesene 188
7.2.4.5. CAGE and granulocyteopenia 188
7.2.4.6. Adenosine and granulocyte adhesion 189

7.3. The studies reported here 189
7.3.1. Do intravascular bubble pass through the cerebral circulation? 190
7.3.2. CBF after CAGE 192
7.3.3. Pial arterial diameter after CAGE 193
7.3.4. CBF, AP, after CAGE 194
7.3.5. Studies with meclofenamate 196
7.3.6. Studies with dextran 500 sulphate 196
7.3.7. Conclusions of the studies performed here 197

7.4. The gas embolism initiated intravascular cell adhesion hypothesis 198
7.4.1. Where are the granulocytes adhering? 199
7.4.2. Complement activation 200
7.4.3. Does CAGE produce an ischemia - reperfusion injury? 201
7.4.4. The formed elements of the blood and CAGE 202
7.4.5. The molecular mechanisms of formed cell mediation of CAGE 202
7.4.5.1. Possible roles of CD11/18 and/or GMP140 203

7.5. Possible future therapies for CAGE 204

7.6. Conclusions and future directions 205

Appendix A. Miscellaneous studies which further characterise the model 207

A.1. Characterisation of the effects of CAGE on the brain 207
A.2. Bubble trapping studies 207
A.2.1. Collection of air from the sagittal sinus 208
A.2.1.1. Surgical preparation for air collection 208
A.2.1.2. Results of air collection studies 209
A.2.2. Ultrasonic Doppler detection of air in the sagittal sinus 209
A.2.2.1. Surgical preparation for ultrasonic Doppler studies 210
A.2.2.2. Results of ultrasonic Doppler bubble trapping studies 210
A.2.3. Conclusions of bubble trapping studies 211
A.3. Cerebral blood flow measured by laser Doppler flowmetry 212
A.3.1 Method used for laser Doppler flowmetry 213
A.3.2.1. Surgical preparation for laser Doppler studies 213
A.3.2.2. Results and conclusions of laser Doppler studies 213
The natural history of air embolism of the brain was studied by observing bubbles in the pial vessels of rabbits and the effect of different doses of air on brain function and blood flow. Air was injected through a cannula placed near the left internal carotid artery, which remained patent throughout the experiment. The smallest amount of intracarotid air that could be seen in the pial vessels was 25 µl. This dose of air passed through the pial vessels rapidly producing a transient pial arteriolar vasodilation which was followed by a progressive reduction of cerebral blood flow and brain electrical activity. There was no effect on right cerebral blood flow demonstrating this insult is limited to the side of injection. This model is thought to correlate well with the natural history of divers with air embolism of the brain.

Various doses of intracarotid air up to 400 µl were also given. A 400 µl dose of intracarotid air produced air embolism in which there was temporary bubble trapping. This was accompanied by a transient pial arterial vasodilation, a progressive reduction in brain blood flow and a sustained deterioration in brain function. All doses of intracarotid air caused:

1. a transient dilation of the pial arteries to as much as 140% of baseline which recovered within 30 to 45 minutes;
2. a progressive reduction of cerebral blood flow to approximately 50% of baseline for the 3 hours of the experiment, and;
3. suppression of the amplitude of the second peak of the somatosensory evoked responses to approximately 40% of baseline. This suppression of evoked responses was progressive for the 3 hours of the experiment in doses of intracarotid air less than 200 µl. A 400 µl dose of air produced a sudden suppression of the second peak of the somatosensory evoked responses to 28%.
of baseline which gradually recovered to approximately 40% of baseline.

Treatment of rabbits with melphalan 3 days prior to experiment reduced the white cell count to 10% of baseline, affecting mainly the granulocyte numbers. When compared to baseline or to untreated controls, leukocytopenia did not change the pial arteriolar response to air embolism. Similarly, cerebral blood flow and somatosensory evoked responses were not significantly affected in the leukocytopenic group given cerebral arterial air embolism.

Further studies in which rabbits were treated with dextran sulphate (m.w. 500,000) to reduce granulocyte adhesion to vascular endothelium showed this treatment also provided significant protection against the effects of cerebral arterial air embolism. Cerebral arterial air embolism induced a sustained pial arterial dilatation to approximately 120% of baseline in the dextran sulphate treated rabbits. Cerebral blood flow increased to 145% of baseline by 15 minutes post embolus but by 30 minutes had recovered to approximately 75% of baseline. Somatosensory evoked responses showed a transient suppression to 50% of baseline before recovering to approximately 75% of baseline for the duration of the study.

A new model for the pathophysiological basis of CAGE is proposed in which the formed elements of the circulation, most likely the granulocytes, adhere to vascular endothelium damaged by passage of air bubbles and further damage adjacent brain. It appears this damage can be largely prevented if granulocytes are inhibited from adhering to the vascular endothelium.