ACUTE AND CHRONIC ATRIAL REMODELING IN OBSTRUCTIVE SLEEP APNOEA: IMPLICATIONS FOR ATRIAL FIBRILLATION

By

Hany R. Dimitri

M.B.B.S, F.R.A.C.P

Department of Cardiology
Royal Adelaide Hospital

&

Discipline of Medicine
University of Adelaide

A thesis submitted to the University of Adelaide in fulfilment of the requirements for the degree of

Doctor of Philosophy

December 2011
Dedicated to

My Mother

My Father

&

My wife Rhiannon
Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABSTRACT</td>
<td>7</td>
</tr>
<tr>
<td>DECLARATION</td>
<td>10</td>
</tr>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>11</td>
</tr>
<tr>
<td>SCHOLARSHIPS DURING CANDIDATURE</td>
<td>15</td>
</tr>
<tr>
<td>PUBLICATIONS AND COMMUNICATIONS TO LEARNED SOCIETIES</td>
<td>16</td>
</tr>
<tr>
<td>ABBREVIATIONS</td>
<td>18</td>
</tr>
<tr>
<td>1. INTRODUCTION &amp; LITERATURE REVIEW</td>
<td>19</td>
</tr>
<tr>
<td>1.1 INTRODUCTION</td>
<td>19</td>
</tr>
<tr>
<td>1.2 OVERVIEW OF THESIS</td>
<td>21</td>
</tr>
<tr>
<td>1.3 ATRIAL FIBRILLATION</td>
<td>22</td>
</tr>
<tr>
<td>1.3.1 A HISTORICAL PERSPECTIVE</td>
<td>22</td>
</tr>
<tr>
<td>1.3.2 EPIDEMIOLOGY OF ATRIAL FIBRILLATION</td>
<td>24</td>
</tr>
<tr>
<td>1.3.3 PATHOPHYSIOLOGY OF ATRIAL FIBRILLATION</td>
<td>29</td>
</tr>
<tr>
<td>1.3.3.1 HISTORICAL OVERVIEW OF MECHANISMS</td>
<td>29</td>
</tr>
<tr>
<td>1.3.3.2 IMPORTANCE OF THE PULMONARY VEINS</td>
<td>35</td>
</tr>
<tr>
<td>1.3.3.3 TRIGGERS, SUBSTRATE AND MODULATORS</td>
<td>37</td>
</tr>
<tr>
<td>1.3.3.3.1 NORMAL ATRIAL ELECTROPHYSIOLOGY</td>
<td>38</td>
</tr>
<tr>
<td>1.3.3.3.2 ANIMAL STUDIES ON ATRIAL REMODELING</td>
<td>39</td>
</tr>
<tr>
<td>1.3.3.3.2.1 ATRIAL FIBRILLATION</td>
<td>39</td>
</tr>
<tr>
<td>1.3.3.3.2.2 HEART FAILURE</td>
<td>46</td>
</tr>
<tr>
<td>1.3.3.3.2.3 AGEING</td>
<td>47</td>
</tr>
<tr>
<td>1.3.3.3.2.4 HYPERTENSION</td>
<td>47</td>
</tr>
<tr>
<td>1.3.3.3.2.5 HYPOXIA, HYPERCAPNIA AND OSA</td>
<td>48</td>
</tr>
<tr>
<td>1.3.3.3.2.6 CORONARY ARTERY DISEASE</td>
<td>50</td>
</tr>
<tr>
<td>1.3.3.3.3 HUMAN STUDIES ON ATRIAL REMODELING</td>
<td>51</td>
</tr>
<tr>
<td>1.3.3.3.3.1 AGE</td>
<td>51</td>
</tr>
<tr>
<td>1.3.3.3.3.2 HYPERTENSION</td>
<td>52</td>
</tr>
<tr>
<td>1.3.3.3.3.3 CONGESTIVE CARDIAC FAILURE</td>
<td>52</td>
</tr>
<tr>
<td>1.3.3.3.3.4 SINUS NODE DISEASE</td>
<td>53</td>
</tr>
<tr>
<td>1.3.3.3.3.5 MITRAL VALVE DISEASE</td>
<td>54</td>
</tr>
<tr>
<td>1.3.3.3.3.6 ATRIAL SEPTAL DEFECT</td>
<td>56</td>
</tr>
<tr>
<td>1.3.3.3.3.7 ATRIAL FIBRILLATION</td>
<td>57</td>
</tr>
<tr>
<td>1.3.3.3.3.8 PACING</td>
<td>58</td>
</tr>
<tr>
<td>1.3.3.3.3.9 PULMONARY VEINS</td>
<td>59</td>
</tr>
<tr>
<td>1.3.3.3.4 COMPLEX ATRIAL SIGNALS</td>
<td>59</td>
</tr>
<tr>
<td>1.3.3.3.5 CATHETER ABLATION OF ATRIAL FIBRILLATION</td>
<td>61</td>
</tr>
<tr>
<td>1.3.3.3.6 MECHANISMS OF STRUCTURAL CHANGE IN AF AND POTENTIAL INTERACTIONS WITH OSA</td>
<td>63</td>
</tr>
<tr>
<td>1.3.3.3.6.1 ATRIAL DILATATION</td>
<td>64</td>
</tr>
<tr>
<td>1.3.3.3.6.2 CELLULAR DEDIFFERENTIATION</td>
<td>65</td>
</tr>
<tr>
<td>1.3.3.3.6.3 APOPTOSIS</td>
<td>66</td>
</tr>
<tr>
<td>1.3.3.3.6.4 FIBROSIS</td>
<td>66</td>
</tr>
<tr>
<td>1.3.3.3.6.5 RENIN-ANGIOTENSION-ALDOSTERONE SYSTEM</td>
<td>67</td>
</tr>
<tr>
<td>1.3.3.3.6.6 TGFβ/SMAD</td>
<td>70</td>
</tr>
<tr>
<td>1.3.3.3.6.7 INFLAMMATION</td>
<td>72</td>
</tr>
<tr>
<td>1.3.3.3.6.7.1 AF</td>
<td>72</td>
</tr>
<tr>
<td>1.3.3.3.6.7.2 OSA</td>
<td>75</td>
</tr>
<tr>
<td>1.3.3.3.6.8 OXIDATIVE STRESS</td>
<td>76</td>
</tr>
</tbody>
</table>
1.3.3.3.6.8.1 AF ................................................................. 77
1.3.3.3.6.8.2 OSA ......................................................... 78
1.3.3.3.6.9 ENDOTHELIAL DYSFUNCTION ......................... 79
1.3.3.3.6.9.1 AF ............................................................. 79
1.3.3.3.6.9.2 OSA .......................................................... 80
1.3.3.3.7 AUTONOMIC NERVOUS SYSTEM ......................... 81
1.3.3.3.7.1 RELEVANCE TO AF ........................................ 81
1.3.3.3.7.2 ANATOMY ..................................................... 82
1.3.3.3.7.3 PARASYMPATHETIC NERVOUS SYSTEM ............... 83
1.3.3.3.7.3.1 AF ............................................................. 83
1.3.3.3.7.3.2 OSA .......................................................... 84
1.3.3.3.7.4 SYMPATHETIC NERVOUS SYSTEM ...................... 84
1.3.3.3.7.4.1 AF ............................................................. 84
1.3.3.3.7.4.2 OSA .......................................................... 85
1.3.3.3.7.5 GANGLIONATED PLEXI .................................. 89
1.3.3.3.7.5.1 AF ............................................................. 89
1.3.3.3.7.5.2 OSA .......................................................... 90
1.3.3.3.7.6 PV-LA JUNCTION ............................................ 90
1.3.3.3.7.7 SUMMARY .................................................... 91

1.4 OBSTRUCTIVE SLEEP APNOEA .................................... 92
1.4.1 HISTORY OF OBSTRUCTIVE SLEEP APNOEA ................. 92
1.4.2 EPIDEMIOLOGY OF OBSTRUCTIVE SLEEP APNOEA ......... 93
1.4.3 EVALUATION OF SLEEP APNOEA ............................ 101
1.4.4 PATHOPHYSIOLOGY OF OBSTRUCTIVE SLEEP APNOEA .... 103
1.4.4.1 NORMAL SLEEP ....................................................... 103
1.4.4.2 CONTRIBUTORS TO OBSTRUCTIVE SLEEP APNOEA ....... 104
1.4.4.2.1 ANATOMICAL CONSIDERATIONS .......................... 105
1.4.4.2.2 MECHANICAL CONSIDERATIONS .......................... 107
1.4.4.2.3 NEUROMUSCULAR CONSIDERATIONS ..................... 108
1.4.4.2.4 VENTILATORY CONTROL STABILITY ...................... 110
1.4.4.2.5 AROUSALS ....................................................... 112
1.4.4.3 PATHOPHYSIOLOGY DUE TO OSA ......................... 112
1.4.4.3.1 CARDIAC STRUCTURAL CHANGES ....................... 112
1.4.4.3.2 AUTONOMIC NERVOUS SYSTEM CHANGES .............. 115
1.4.4.3.2.1 CENTRAL PROCESSES .................................... 116
1.4.4.3.2.2 LUNG REFLEXES .......................................... 117
1.4.4.3.2.3 CARDIAC REFLEXES ...................................... 117
1.4.4.3.2.4 INTRATHORACIC PRESSURE ............................. 118
1.4.4.3.2.5 HYPOXIA AND HYPERCAPNIA .......................... 118
1.4.5 ADVERSE CLINICAL CONSEQUENCES OF OBSTRUCTIVE SLEEP APNOEA .................................................. 119
1.4.5.1 HYPERTENSION ..................................................... 119
1.4.5.2 INSULIN RESISTANCE, DIABETES AND DYSLIPIDAEMIA .... 122
1.4.5.3 CORONARY ARTERY DISEASE ................................. 123
1.4.5.4 STROKE ............................................................. 125
1.4.5.5 CARDIOVASCULAR MORTALITY ............................. 125
1.4.5.6 SUDDEN CARDIAC DEATH ...................................... 126
1.4.5.7 ATRIAL FIBRILLATION ............................................ 127
1.4.6 ASSOCIATION OF AF AND CENTRAL SLEEP APNOEA ....... 131

1.5 RATIONALE FOR THIS THESIS AND AIMS ...................... 134

2 ATRIAL SUBSTRATE REMODELING IN PAROXYSMAL AND PERSISTENT
ATRIAL FIBRILLATION: SIGNAL FRAGMENTATION AND VOLTAGE 135
2.1 INTRODUCTION .......................................................... 135
2.2 METHODS ........................................................................ 136
## REFERENCES

4.3.3.4 SINUS NODE FUNCTION .................................................. 182
4.3.3.5 ELECTROANATOMIC MAPPING ...................................... 182
4.3.3.6 VOLTAGE ABNORMALITIES .......................................... 182
4.3.3.7 CONDUCTION ABNORMALITIES ..................................... 183
4.3.3.8 COMPLEX FRACTIONATED ELECTROGRAMS ....................... 183

## DISCUSSION

4.4 MAJOR FINDINGS .............................................................. 184
4.4.1 MAJOR FINDINGS ............................................................ 184
4.4.2 COMMENT ...................................................................... 185
4.4.3 LIMITATIONS ................................................................. 189

## CONCLUSION

4.5 CONCLUSION .................................................................... 190

### NOCTURNAL ALTERATIONS OF ATRIAL ELECTROPHYSIOLOGY IN OBSTRUCTIVE SLEEP APNOEA: EVIDENCE FOR ACUTE ELECTRICAL REMODELING

5.1 INTRODUCTION ................................................................ 197
5.2 METHODS ................................................................. 198
5.2.1 SLEEP STUDY ............................................................... 198
5.2.2 ABLATION PROCEDURE ................................................... 200
5.2.3 POST-PROCEDURE STUDY PROTOCOL ............................. 201
5.2.4 STATISTICAL ANALYSIS .................................................. 203

## RESULTS

5.3 POLYSOMNOGRAPHIC DATA ............................................... 204
5.3.1 POLYSOMNOGRAPHIC DATA ............................................ 204
5.3.2 EFFECTIVE REFRACTORY PERIOD .................................... 205
5.3.3 CONDUCTION TIME AND CONDUCTION DELAY ............... 206

## DISCUSSION

5.4 MAJOR FINDINGS .............................................................. 208
5.4.1 MAJOR FINDINGS ............................................................ 208
5.4.2 COMMENT ...................................................................... 209
5.4.3 LIMITATIONS ................................................................. 216

## CONCLUSION

5.5 CONCLUSION .................................................................... 218

### THE ACUTE EFFECTS OF HYPOXIA AND HYPERCAPNIA ON ATRIAL ELECTROPHYSIOLOGY: AN EX-VIVO RABBIT MODEL

6.1 INTRODUCTION ............................................................. 226
6.2 METHODS ...................................................................... 227
6.2.1 SPECIMEN PREPARATION ................................................ 227
6.2.2 EXPERIMENTAL DESIGN .................................................. 227
6.2.3 MODEL VALIDATION ....................................................... 229
6.2.4 ELECTROPHYSIOLOGY STIMULATION PROTOCOL ............ 229
6.2.5 STATISTICAL ANALYSIS .................................................. 230

## RESULTS

6.3 MODEL VALIDATION .......................................................... 231
6.3.1 MODEL VALIDATION ....................................................... 231
6.3.2 ATRIAL REFRACTORINESS .............................................. 231
6.3.3 ATRIAL TISSUE CONDUCTION VELOCITY AND HETEROGENEITY .................................................. 233

## DISCUSSION

6.4 MAJOR FINDINGS .............................................................. 235
6.4.1 MAJOR FINDINGS ............................................................ 235
6.4.2 COMMENT ...................................................................... 236
6.4.3 LIMITATIONS ................................................................. 240

## CONCLUSION

6.5 CONCLUSION .................................................................... 242

### SUMMARY

FUTURE DIRECTIONS .................................................................. 250
REFERENCES ........................................................................ 256

REFERENCES ........................................................................ 262
ABSTRACT

Atrial fibrillation (AF), the most common sustained arrhythmia, has been well studied; however, its underlying mechanisms and relationships to other disease processes have not been fully explored. Observational data from epidemiological studies have suggested a relationship between obstructive sleep apnoea (OSA) and AF. Recent clinical studies have implicated an adverse outcome to therapy in patients with AF and OSA. Despite several candidate mechanisms advanced, the acute and chronic changes to the atrial myocardium have not been fully characterised.

This thesis evaluates symptomatic patients with AF presenting for radiofrequency ablation of their arrhythmia. The acute and chronic electrophysiological and electroanatomical atrial substrate is characterised. Finally the effect on clinical outcomes of therapy directed at AF is evaluated with specific reference to the presence of OSA.

Chapter 2 demonstrates the differences in the atrial substrate in paroxysmal and persistent AF. This study found persistent AF was associated with a reduction in electrogram voltage and greater signal fragmentation, with these two attributes being negatively associated.

Chapter 3 examines patients presenting with symptomatic AF, previously not known to have sleep disordered breathing. OSA was associated with a greater symptomatic burden of AF as well as chronicity of the arrhythmia. In the presence of more severe OSA, there was a greater chance of failure of radiofrequency ablation in maintaining sinus rhythm.
Chapter 4 characterises the underlying atrial substrate resulting in AF in patients with moderate to severe untreated OSA. OSA patients had larger atria, greater areas of low voltage and areas of electrical silence, suggesting loss of atrial myocardium. There were also markedly reduced conduction velocities, longer corrected sinus node recovery times and more conduction abnormalities characterised by complex electrograms. These findings provide important insights into the adverse remodeling that may allow AF to develop and persist in these patients, and promote the failure of ablation strategies.

Chapter 5 examines nocturnal atrial electrophysiological alterations resulting from acute episodes of respiratory disturbance associated with hypopnoea and obstructive apnoea events the night after undergoing radiofrequency ablation for AF. Dynamic changes in effective refractory period (ERP), conduction times and conduction delay along linear catheters were documented. The changes appeared to be more marked for obstructive apnoeas than hypopnoeas. This study suggests a dynamic, pro-arrhythmic electrical substrate that could potentially lead to nocturnal triggering of AF and its maintenance.

Finally, Chapter 6 describes an ex-vivo rabbit model, created to examine the acute effects of hypoxia (moderate and severe) and hypercapnia. Left atrial ERP, conduction time and conduction heterogeneity were studied using a customised microelectrode array. During hypoxia, there was a dose dependent increase in ERP with only partial resolution on restoration of baseline oxygen levels. With hypercapnia, there was a slower rise in ERP that did not appear to recover. Slowing of conduction was most noted in severe hypoxia, with
only partial resolution in recovery. In hypercapnia, there was progressive slowing of conduction into recovery. Conduction heterogeneity was also increased in the presence of hypoxia and hypercapnia. These findings further suggest complex alterations to the atrial electrophysiology in the presence of hypoxia and hypercapnia, with dynamic changes to in refractory periods, conduction times and heterogeneity.

Together, a greater understanding of the acute and chronic effects of OSA on AF, and an appreciation of the failure of ablation strategies in the presence of this breathing disorder is attained. This paves the way for further studies on the mechanisms involved, and on the potential role for continuous positive airway pressure therapy (CPAP) in the management of patients with OSA and AF.
DECLARATION

This work was performed by the candidate at the Centre for Heart Rhythm Disorders at the University of Adelaide and Royal Adelaide Hospital, South Australia (during the years 2007 through 2011).

This work contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text.

I give consent to this copy of my thesis, when deposited in the University Library, being made available for loan and photocopying, subject to the provisions of the Copyright Act 1968. The author acknowledges that copyright of published works derived from this thesis (specifically, chapter 4) resides with the copyright holder of those works.


Hany R. Dimitri,
Department of Cardiology,
Royal Adelaide Hospital.
&
Discipline of Medicine
University of Adelaide,
Adelaide, South Australia.
December 2011
ACKNOWLEDGEMENTS

I would like to thank Professor Prashanthan Sanders, my primary supervisor and mentor, for the opportunity to complete this PhD. I would also like to thank Dr Glenn Young, my secondary supervisor, for his patient help, particular with the collection of data. It has been a privilege, pleasure and honour to work with this internationally acclaimed group of doctors, scientists and auxiliary staff. The group as a whole, under the leadership of Professor Sanders, have a most admirable dedication to research and furthering knowledge in cardiac arrhythmias, with a special focus on atrial fibrillation.

I am especially grateful to Professor Ian Wilcox for introducing me to the world of obstructive sleep apnoea and its cardiovascular associations, as well as awakening my mind to the wonders of medical research. It was his constant reinforcement of the importance of this breathing condition, especially its association with atrial fibrillation, that encouraged me to pursue further studies in the area. I am thankful for his encouragement and continued mentorship.

During the writing up period of this work, I was supported, encouraged and guided by Associate Professor David Richards. I am extremely grateful for his extraordinary patience, understanding and counselling, without which this project would have been more difficult to complete.

I am grateful for the financial support by means of research grants from the Cardiac Society of Australia (year one), and the Heart Foundation and National Health and Medical Research Council (years two and three). I am eternally grateful to the patients who agreed to
participate in these studies, in particular, those who endured extra procedural time to allow collection of data. I am most grateful to those select patients who agreed to participate in an overnight evaluation of their atrial electrophysiology as this necessitated a full night of intervention after their ablation procedure.

I am thankful to my colleagues and in particular, Fellows in the Electrophysiology department of the Royal Adelaide Hospital. It was an absolute delight to work with and enjoy the support of Martin Stiles, Bobby John, Dennis Lau, Gautam Sharma, Narayanan Namboodiri, Darryl Leong, Hany Abed, Muayad Alasady, and Han Lim. I must especially mention Dr Mitra Masoumeh for assisting with the collection of echocardiographic data. Special mention goes to Dr Anthony Brooks for his hard work and superlative knowledge, especially in the area of statistics. I am incredibly grateful to Dr Pawel Kuklik, in particular, for providing support in computer programming to the groups, and for creating the software that was instrumental in the analysis for these studies. On this note, I cannot thank enough Mr Bruce Lobb for his major contribution to my animal study by creating the custom made microelectrode array – without both of their contributions, this thesis could not be completed. I would also like to thank Mr Christopher Wong, a medical student with a superb drive for collection and understanding of data.

My thanks also extend to the administrative staff, importantly, Melissa, Linda and Aimie. Melissa, a loyal and dedicated personal assistant to Professor Sanders, was instrumental in the management of clinical studies in the department, and the collection of data, dedicating
her time and efforts to duties to ensure we had access to clinical information necessary for keeping databases up to date.

Given the topic of this thesis, the input, guidance and assistance from the Royal Adelaide Hospital and Repatriation Hospital Department of Thoracic medicine, is gratefully acknowledged. I would like to specifically thank Drs Ral and Nick Antic, Dr Andrew Thornton and Professor Doug McEvoy for their expert tuition and guidance on the ins and outs of sleep apnoea. I cannot thank them enough for the abundant time and energy dedicated to scoring the seemingly hundreds of sleep studies performed. In particular, my thanks to sleep technologists Michelle Ng, Joel Pannell and Alison Fitch for dedicating their time to providing timely polysomnography reports.

The supervision of Dr David Saint was pivotal for the completion of the animal experiment, which is the basis of a major chapter of this thesis. I had essentially no prior experience in working with animals, and I could not have completed this component of my research without Dr Saint’s expert advice and guidance. I appreciate the dedication and assistance of Melissa Neo and Julia Kim from Dr Saint’s laboratory, in setting up my experiments, caring for the rabbits and helping me collect the data.

During the time of this thesis, my dearly loved mother passed away. It was my mother’s dedication to my education that propelled me into university and my medical career. I am forever grateful for her support, encouragement, persistence and, most of all, love. Without her upbringing, I would not have made it to this privileged position. I must also thank my father for his wonderful kindness and patience with me,
especially through these difficult times. It was through the love and dedication of my parents that I was empowered to learn, achieve and ultimately succeed. God Bless you both.

Lastly, I cannot thank enough my wife Rhiannon. I am truly fortunate, and I have survived only with her everlasting and tireless support throughout this stressful time. Rhiannon's dedication and love of our son Marcus allowed me the necessary time to complete this work. Her kindness, patience, understanding and help I can never repay.
SCHOLARSHIPS DURING CANDIDATURE

2007
Postgraduate Scholarship from the Cardiac Society of Australia and New Zealand scholarship

2008 & 2009
Medical Postgraduate Scholarship, jointly funded by the National Heart Foundation of Australia, and, the National Health and Medical Research Council of Australia
PUBLICATIONS AND COMMUNICATIONS TO LEARNED SOCIETIES

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HEART RHYTHM SOCIETY SCIENTIFIC SESSIONS 2011
Hany Dimitri, Michelle Ng, Pawel Kuklik, Anthony Brooks, Martin Stiles, Andrew Thornton, Ral Antic, Prashanthan Sanders. Chronic obstructive sleep apnoea causes atrial remodeling: Implications for atrial fibrillation. Heart Rhythm 2011;8(5):S244
PUBLICATION:


CHAPTER 5:
ABSTRACT POSTER PRESENTATION: CARDIAC SOCIETY OF AUSTRALIA AND NEW ZEALAND SCIENTIFIC SESSIONS 2010
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CHAPTER 6:
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### ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
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<tr>
<td>AF</td>
<td>atrial fibrillation</td>
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<tr>
<td>AHI</td>
<td>apnoea hypopnoea index</td>
</tr>
<tr>
<td>CFAE</td>
<td>complex fractionated atrial electrograms</td>
</tr>
<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
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<tr>
<td>CSA</td>
<td>central sleep apnoea</td>
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<tr>
<td>CSNRT</td>
<td>corrected sinus node recovery time</td>
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</tbody>
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