



Matt Johnson  
Leanne Boyd  
Hugh Grantham and  
Kathryn Eastwood

# PARAMEDIC PRINCIPLES AND PRACTICE ANZ

A clinical reasoning approach

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**Matt Johnson**

BAPPSci, DIPAMBSTUDIES, GRADDIPEMERGHEALTH,  
GRADCERTHEALTHPROFED, MEMERG HEALTH, FPA

**Leanne Boyd**

DIPAPPSci, BNURS, GRADCERTCRITCARE,  
GRADCERTHIGHERED, MNURS, MTEM, PHD

**Hugh Grantham**

ASM, MBBS, FRACGP

**Kathryn Eastwood**

RN, BSc, BNURS, BPARAMEDSTUDIES,  
DIPAMBPARASTUDIES, GRADDIPEMERGHlth (MICA),  
GRADCERTHIGHERED, MEMERGHEALTH (MICA),  
PHD CANDIDATE

# Contents

Foreword	xvii	Medicine vs paramedicine?	34
Preface	xviii	Using a structured approach to combat complexity	36
About the authors	xix	The emergency model	36
Contributors	xx	The medical interview model	40
Reviewers	xxiii	<b>Chapter 5: The clinical reasoning process</b>	<b>43</b>
The key to improving your clinical practice	xxiv	Introduction	43
Acknowledgements	xxvi	Case study 1	44
<b>Chapter 1: Introduction</b>	<b>1</b>	Clinical decision making in the face of uncertainty: the paramedic paradigm	44
Paramedic principles and practice	1	Problem solving versus decision making	45
Essential knowledge	6	The myth of the expert	46
Summary	6	Developing clinical reasoning skills	49
<b>PART 1</b>	<b>7</b>	A step-by-step guide to clinical decision making	53
<b>PARAMEDIC PRINCIPLES</b>	<b>7</b>	Algorithms and cognitive checks	54
<b>SECTION 1: PRINCIPLES OF PARAMEDIC PRACTICE</b>	<b>8</b>	A syndrome approach to patient management	55
<b>Chapter 2: The paramedic role in healthcare</b>	<b>8</b>	Error wisdom	57
Introduction	9	Summary	57
Case study 1	9	<b>Chapter 6: The patient interview</b>	<b>59</b>
The role of ambulance services in Australia and New Zealand	10	Introduction	59
Professionalism and professional standards	14	The structured patient interview	60
Terminology and qualifications	15	The science and art of communication	61
The future paramedic role	16	The paramedic interview setting	65
Summary	19	Paramedic–patient interview structure	65
<b>Chapter 3: Characteristics of ambulance patients</b>	<b>21</b>	Case study 1	65
Introduction	21	Barriers to effective communication	74
Who are ambulance patients?	21	Summary	76
From symptom onset to the decision to call an ambulance	24	<b>SECTION 3: PATIENT AND PARAMEDIC SAFETY</b>	<b>78</b>
From calling for an ambulance to ambulance arrival	27	<b>Chapter 7: Patient safety and paramedicine</b>	<b>78</b>
Summary	28	Introduction	79
<b>SECTION 2: THE PARAMEDIC'S CLINICAL APPROACH</b>	<b>32</b>	The harm caused by healthcare errors	79
<b>Chapter 4: The structured clinical approach</b>	<b>32</b>	Types of medical error	80
Introduction	33	Models of error	80
Case study 1	33	Reducing diagnostic errors	81
		Error defence	81
		Error management	82
		Case study 1	83
		Evidence-based practice and patient safety	84
		EBP, individual patients and clinical reasoning	86
		Summary	86

## Chapter 8: Paramedic health and wellbeing

Introduction	88
Wellbeing	88
Paramedic health and safety	88
Stress	89
Managing emotions	91
Case study 1	91
Fatigue	93
Case study 2	93
Shift work	94
Occupational violence	95
Case study 3	95
Injury	97
Case study 4	97
Student paramedics	98
Getting help	100
Summary	101

## SECTION 4: PARAMEDIC EDUCATION 103

### Chapter 9: Paramedic education 103

Introduction	104
Experience versus education	104
The history of paramedic education in Australasia	105
Case study 1	105
International educational standards	107
Paramedic education in the university setting	107
Course accreditation and professional regulation	109
Continuing professional development and postgraduate education	110
Educational challenges and future directions	111
Summary	112

## SECTION 5: LEGAL AND ETHICAL CONSIDERATIONS 114

### Chapter 10: Legal and ethical considerations in clinical decision making 114

Introduction	115
Ethics and the law	115
Case study 1	115
Consent	119
Refusal of treatment	120
Elements of consent	121
Case study 1 evaluation	124
Documentation	125
Case study 2	125
1. Assess	125
2. Confirm	126
3. Treat	126
4. Evaluate	127
Summary	127

## SECTION 6: CLINICAL REASONING AND THE PARAMEDIC MODEL OF PRACTICE 128

### Chapter 11: Developing a philosophy of practice 128

Introduction	129
Models of practice	129
Case study 1	129
Building a philosophy of practice	133
Summary	135

## PART 2 PARAMEDIC PRACTICE 137

### SECTION 7: THE PARAMEDIC APPROACH TO THE PATIENT IN AN ALTERED CONSCIOUS STATE 138

#### Chapter 12: Hypoglycaemia 139

Introduction	139
Pathophysiology	141
Management of blood glucose	143
Case study 1	146
1. Assess	146
2. Confirm	148
3. Treat	151
4. Evaluate	154
At the hospital ED	155
Hospital admission	155
Discharge criteria	155
Hypoglycaemia across the lifespan: paediatric considerations	156
Case study 2	157
1. Assess	157
2. Confirm	157
3. Treat	158
4. Evaluate	159
Future research	159
Summary	159

#### Chapter 13: Cerebrovascular accidents 161

Introduction	161
Pathophysiology	161
Clinical manifestations	164
Case study 1	167
1. Assess	167
2. Confirm	172
3. Treat	174
4. Evaluate	176
Ongoing management	177
Investigations	177
Hospital admission	177
Stroke across the lifespan	177
Case study 2	178
1. Assess	179



2. Confirm	179	SECTION 8: THE PARAMEDIC	
3. Treat	180	APPROACH TO THE PATIENT	
4. Evaluate	182	IN RESPIRATORY DISTRESS	226
Case study 3	182	Chapter 16: Airway obstruction	227
1. Assess	183	Introduction	228
2. Confirm	183	Pathophysiology	228
3. Treat	184	Case study 1	230
4. Evaluate	185	1. Assess	230
Case study 4	186	2. Confirm	232
1. Assess	187	3. Treat	232
2. Confirm	187	4. Evaluate	233
3. Treat	187	Ongoing management	234
4. Evaluate	187	Hospital admission	234
Future research	188	Follow-up	234
Summary	189	Case study 2	234
Chapter 14: Overdose	189	1. Assess	235
Introduction	189	2. Confirm	235
Pathophysiology	195	3. Treat	235
Case study 1	195	4. Evaluate	236
1. Assess	197	Case study 3	236
2. Confirm	199	1. Assess	236
3. Treat	200	2. Confirm	237
4. Evaluate	201	3. Treat	238
Ongoing treatment	202	4. Evaluate	238
Investigations	202	Summary	238
Hospital management	202	Chapter 17: Asthma	240
Case study 2	203	Introduction	240
1. Assess	203	Pathophysiology	240
2. Confirm	205	Case study 1	245
3. Treat	206	1. Assess	245
4. Evaluate	206	2. Confirm	249
Case study 3	207	3. Treat	250
1. Assess	207	4. Evaluate	253
2. Confirm	208	Ongoing management	254
3. Treat	208	Follow-up	255
4. Evaluate	208	Long-term impact	255
Future research	208	Case study 2	255
Summary	211	1. Assess	255
Chapter 15: Seizures	211	2. Confirm	256
Introduction	211	3. Treat	256
Pathophysiology	213	4. Evaluate	258
Seizure classification	215	Future research	258
Management	216	Summary	259
Case study 1	216	Chapter 18: Acute pulmonary oedema	260
1. Assess	218	Introduction	260
2. Confirm	220	Pathophysiology	260
3. Treat	222	Case study 1	264
4. Evaluate	222	1. Assess	264
Ongoing management	222	2. Confirm	265
Hospital admission	223	3. Treat	267
Case study 2	223	4. Evaluate	269
1. Assess	224	Hospital admission	269
2. Confirm	224	Long-term impact	270
3. Treat	225	Acute pulmonary oedema across the lifespan	270
4. Evaluate			
Summary			

Case study 2	270	Chapter 21: Pulmonary embolism	313
1. Assess	270	Introduction	313
2. Confirm	270	Pathophysiology	313
3. Treat	272	Risk factors	316
4. Evaluate	272	Case study 1	317
Case study 3	273	1. Assess	317
1. Assess	273	2. Confirm	319
2. Confirm	273	3. Treat	321
3. Treat	274	4. Evaluate	322
4. Evaluate	274	Ongoing management	322
Ongoing management	274	Investigations	322
Case study 4	275	Hospital admission	323
1. Assess	275	Follow-up	323
2. Confirm	275	Case study 2	323
3. Treat	276	1. Assess	323
4. Evaluate	276	2. Confirm	324
Ongoing management	277	3. Treat	325
Future research	277	4. Evaluate	325
Summary	277	Research	326
Chapter 19: Chronic obstructive pulmonary disease	279	Summary	326
Introduction	279	Chapter 22: Pleural effusion	327
Pathophysiology	280	Introduction	327
Case study 1	285	Pathophysiology	327
1. Assess	285	Case study 1	330
2. Confirm	288	1. Assess	330
3. Treat	290	2. Confirm	332
4. Evaluate	293	3. Treat	334
Hospital admission	294	4. Evaluate	334
Investigations	295	Ongoing management	335
Follow-up	295	Investigations	335
Case study 2	296	Hospital admission	335
1. Assess	296	Follow-up	335
2. Confirm	297	Case study 2	336
3. Treat	298	1. Assess	336
4. Evaluate	299	2. Confirm	336
Future research	300	3. Treat	337
Summary	300	4. Evaluate	338
Chapter 20: Pneumothorax	301	Summary	338
Introduction	301	Chapter 23: The paediatric patient with a noisy airway	339
Pathophysiology	301	Introduction	339
Case study 1	305	Pathophysiology	339
1. Assess	305	Case study 1	343
2. Confirm	307	1. Assess	343
3. Treat	308	2. Confirm	345
4. Evaluate	309	3. Treat	345
Ongoing management	309	4. Evaluate	346
Hospital admission	310	Ongoing management	347
Long-term impact	310	Hospital admission	347
Case study 2	310	Long-term impact	348
1. Assess	310	Case study 2	348
2. Confirm	310	1. Assess	348
3. Treat	311	2. Confirm	349
4. Evaluate	312	3. Treat	349
Summary	312	4. Evaluate	350

Hospital management	350	2. Confirm	398
Case study 3	350	3. Treat	400
1. Assess	351	4. Evaluate	401
2. Confirm	351	Case study 3	401
3. Treat	352	1. Assess	401
4. Evaluate	352	2. Confirm	402
Future research	352	3. Treat	403
Summary	352	4. Evaluate	404
		Summary	404
<b>SECTION 9: THE PARAMEDIC APPROACH TO THE PATIENT SUFFERING A CARDIAC EMERGENCY</b>	<b>354</b>	<b>Chapter 26: Cardiac arrest</b>	<b>405</b>
<b>Chapter 24: Chest pain</b>	<b>355</b>	Introduction	405
Introduction	356	Chain of survival	406
Pathophysiology	356	Pathophysiology	407
Myocardial perfusion and myocardial workload	359	Case study 1	410
Case study 1	360	1. Assess	410
1. Assess	361	2. Confirm	412
2. Confirm	367	3. Treat	413
3. Treat	371	4. Evaluate	415
4. Evaluate	373	Ongoing management	416
Investigations	374	Long-term outcomes	419
Ongoing management	375	Case study 2	419
Hospital admission	376	1. Assess	419
Follow-up	376	2. Confirm	419
Acute coronary syndrome across the lifespan	376	3. Treat	422
Case study 2	378	4. Evaluate	422
1. Assess	378	Future research	423
2. Confirm	379	Summary	423
3. Treat	380	<b>SECTION 10: THE PARAMEDIC APPROACH TO THE PATIENT WITH A SEVERE ALLERGIC REACTION</b>	<b>426</b>
4. Evaluate	380	<b>Chapter 27: Anaphylaxis</b>	<b>427</b>
Case study 3	380	Introduction	427
1. Assess	381	Pathophysiology	427
2. Confirm	381	Case study 1	430
3. Treat	382	1. Assess	431
4. Evaluate	382	2. Confirm	435
Future research	382	3. Treat	437
Summary	383	4. Evaluate	439
<b>Chapter 25: Arrhythmias</b>	<b>385</b>	Investigations	439
Introduction	385	Ongoing management	439
Pathophysiology	385	Hospital admission	440
Case study 1	390	Follow-up	440
1. Assess	390	Long-term role	440
2. Confirm	393	Anaphylaxis across the lifespan	440
3. Treat	393	Case study 2	444
4. Evaluate	395	1. Assess	444
Ongoing management	395	2. Confirm	445
Hospital admission	396	3. Treat	445
Arrhythmias across the lifespan	396	4. Evaluate	446
Wolff-Parkinson-White	397	Case study 3	446
Long QT syndrome	397	1. Assess	447
Commotio cordis	397	2. Confirm	447
Case study 2	398	3. Treat	448
1. Assess	398	4. Evaluate	449

Case study 4	449	2. Confirm	500
1. Assess	450	3. Treat	500
2. Confirm	450	4. Evaluate	500
3. Treat	450	Future research	500
4. Evaluate	451	Summary	501
Future research	452		
Summary	452		
<b>SECTION 11: THE PARAMEDIC APPROACH TO THE PATIENT PRESENTING WITH PAIN</b>	<b>454</b>	<b>Chapter 30: Renal colic</b>	<b>502</b>
<b>Chapter 28: Pain</b>	<b>455</b>	Introduction	502
Introduction	456	Pathophysiology	502
Pathophysiology	456	Case study 1	504
Case study 1	460	1. Assess	504
1. Assess	461	2. Confirm	505
2. Confirm	465	3. Treat	506
3. Treat	466	4. Evaluate	506
4. Evaluate	468	Hospital emergency management	507
Ongoing management	468	Investigations	507
Case study 2	468	Ongoing management	507
1. Assess	468	Hospital admission	508
2. Confirm	469	Follow-up	508
3. Treat	470	Long-term role	508
4. Evaluate	470	Renal stones across the lifespan	508
Ongoing management	470	Future research	508
Case study 3	470	Summary	508
1. Assess	470		
2. Confirm	471	<b>SECTION 12: THE PARAMEDIC APPROACH TO THE TRAUMA PATIENT</b>	<b>510</b>
3. Treat	472	<b>Chapter 31: The structured clinical approach to trauma patients</b>	<b>511</b>
4. Evaluate	472	Introduction	511
Future research	472	Case study 1	512
Summary	472	Assessment of the trauma patient	512
		Summary	517
<b>Chapter 29: Lower back pain</b>	<b>476</b>	<b>Chapter 32: Head injuries</b>	<b>518</b>
Introduction	476	Introduction	518
Pathophysiology	476	Anatomy	519
Case study 1	481	Pathophysiology	524
1. Assess	481	Case study 1	533
2. Confirm	487	1. Assess	534
3. Treat	490	2. Confirm	538
4. Evaluate	490	3. Treat	539
Ongoing management	491	4. Evaluate	541
Follow-up	493	Ongoing management	542
Outcomes	493	Investigations	542
Back pain across the lifespan	493	Hospital admission	542
Case study 2	494	Hospital discharge	542
1. Assess	494	Head injuries across the lifespan	543
2. Confirm	495	Case study 2	544
3. Treat	496	1. Assess	544
4. Evaluate	496	2. Confirm	544
Case study 3	498	3. Treat	545
1. Assess	498	4. Evaluate	547
2. Confirm	498	Future research	548
3. Treat	499	Summary	548
4. Evaluate	499		
Case study 4	499		
1. Assess	499		



## Chapter 33: Chest injuries

Introduction	550
Pathophysiology	550
Case study 1	560
1. Assess	560
2. Confirm	561
3. Treat	562
4. Evaluate	563
Ongoing management	563
Investigations	563
Hospital admission	563
Long-term role	564
Chest trauma across the lifespan	564

### Case study 2

1. Assess
2. Confirm
3. Treat
4. Evaluate

### Case study 3

1. Assess
2. Confirm
3. Treat
4. Evaluate

Future research

Summary

## Chapter 34: Musculoskeletal injuries

Introduction	571
Pathophysiology	571
Case study 1	577
1. Assess	577
2. Confirm	583
3. Treat	583
4. Evaluate	590
Ongoing management	590
Investigations	591
Ongoing management of uncomplicated injuries	592
Torn muscle repair	592
Management of specific fractures	594
Complications of musculoskeletal injuries	596
Rehabilitation	598
Long-term impact	598

### Case study 2

1. Assess
2. Confirm
3. Treat
4. Evaluate

### Case study 3

1. Assess
2. Confirm
3. Treat
4. Evaluate

Future research

Summary

## Chapter 35: Traumatic spinal injuries

Introduction	606
Pathophysiology	606

550	Case study 1	609
550	1. Assess	610
550	2. Confirm	617
560	3. Treat	618
560	4. Evaluate	627
561	Hospital admission	628
562	Long-term issues and care	629
563	Life expectancy for SCI survivors	630
563	Case study 2	630
563	1. Assess	630
563	2. Confirm	631
564	3. Treat	631
564	4. Evaluate	632
565	Case study 3	632
565	1. Assess	632
565	2. Confirm	633
566	3. Treat	634
567	4. Evaluate	634
568	Future research	634
568	Summary	635
569	Chapter 36: Burns	637
569	Introduction	637
570	Pathophysiology	638
570	'Cold burns'	645
571	Case study 1	645
571	1. Assess	645
571	2. Confirm	648
571	3. Treat	649
577	4. Evaluate	653
577	Hospital admission (burns unit)	654
583	Investigations	655
583	Ongoing management	655
590	Follow-up	656
590	Burns across the lifespan: elderly patients	656
591	Case study 2	656
592	1. Assess	657
592	2. Confirm	657
594	3. Treat	658
596	4. Evaluate	660
598	Paediatric airway burns	660
598	Case study 3	661
599	1. Assess	661
599	2. Confirm	662
599	3. Treat	663
600	4. Evaluate	665
600	Case study 4	665
601	1. Assess	665
601	2. Confirm	667
601	3. Treat	668
602	4. Evaluate	669
602	To intubate or not	669
603	Case study 5	670
603	1. Assess	670
606	2. Confirm	670
606	3. Treat	671
606	4. Evaluate	672

Future research	672	2. Confirm	705
Summary	673	3. Treat	705
		4. Evaluate	706
<b>SECTION 13: THE PARAMEDIC APPROACH TO THE PATIENT PRESENTING WITH ENVIRONMENTAL INJURY</b>	<b>675</b>	Summary	706
<b>Chapter 37: Hypothermia</b>	<b>676</b>	<b>Chapter 40: Snake bites</b>	<b>707</b>
Introduction	676	Introduction	707
Pathophysiology	677	Pathophysiology	707
<b>Case study 1</b>	<b>678</b>	<b>Case study 1</b>	<b>710</b>
1. Assess	679	1. Assess	711
2. Confirm	679	2. Confirm	712
3. Treat	681	3. Treat	712
4. Evaluate	681	4. Evaluate	714
Ongoing management	682	Ongoing management	714
<b>Case study 2</b>	<b>682</b>	Hospital management	715
1. Assess	682	Investigations	717
2. Confirm	683	Hospital admission	717
3. Treat	683	Long-term impact	718
4. Evaluate	683	Snake bites across the lifespan	718
Future research	684	<b>Case study 2</b>	<b>718</b>
Summary	684	1. Assess	719
		2. Confirm	719
<b>Chapter 38: Hyperthermia</b>	<b>685</b>	3. Treat	720
Introduction	685	4. Evaluate	720
Pathophysiology	685	<b>Case study 3</b>	<b>721</b>
<b>Case study 1</b>	<b>687</b>	1. Assess	721
1. Assess	688	2. Confirm	721
2. Confirm	689	3. Treat	722
3. Treat	690	4. Evaluate	722
4. Evaluate	690	Future research	722
Ongoing management	691	Summary	722
Hospital admission	691	<b>Chapter 41: Spider bites</b>	<b>723</b>
Investigations	691	Introduction	723
Follow-up and long-term impact	691	Pathophysiology	723
Hyperthermia across the lifespan	691	<b>Case study 1</b>	<b>725</b>
<b>Case study 2</b>	<b>691</b>	1. Assess	725
1. Assess	692	2. Confirm	727
2. Confirm	692	3. Treat	727
3. Treat	693	4. Evaluate	728
4. Evaluate	693	Ongoing hospital management	728
Summary	694	Hospital admission	728
		Long-term impact	728
<b>Chapter 39: Decompression injuries</b>	<b>695</b>	Spider bites across the lifespan	728
Introduction	695	<b>Case study 2</b>	<b>729</b>
Pathophysiology	695	1. Assess	729
<b>Case study 1</b>	<b>698</b>	2. Confirm	729
1. Assess	698	3. Treat	730
2. Confirm	700	4. Evaluate	731
3. Treat	701	<b>Case study 3</b>	<b>731</b>
4. Evaluate	703	1. Assess	731
Ongoing management	703	2. Confirm	732
Hospital admission	704	3. Treat	732
Long-term impact	704	4. Evaluate	732
<b>Case study 2</b>	<b>704</b>	Summary	732
1. Assess	704	<b>Chapter 42: Marine envenomation</b>	<b>733</b>
		Introduction	733
		Pathophysiology	733

Case study 1	739	Case study 5	772
1. Assess	740	1. Assess	772
2. Confirm	741	2. Confirm	773
3. Treat	742	3. Treat	773
4. Evaluate	742	4. Evaluate	773
Hospital management	743	Case study 6	774
Long-term impact	743	1. Assess	774
Envenomation across the lifespan	743	2. Confirm	774
Case study 2	743	3. Treat	775
1. Assess	743	4. Evaluate	775
2. Confirm	744	Ongoing management	775
3. Treat	744	Bowel obstruction across the lifespan	775
4. Evaluate	745	Summary	776
Hospital management	745	Chapter 44: Sepsis	778
Case study 3	746	Introduction	778
1. Assess	746	Pathophysiology	778
2. Confirm	746	Case study 1	784
3. Treat	747	1. Assess	784
4. Evaluate	747	2. Confirm	787
Future research	748	3. Treat	788
Summary	748	4. Evaluate	791
		Ongoing management	791
SECTION 14: THE PARAMEDIC		Hospital admission	792
APPROACH TO THE UNWELL		Sepsis across the lifespan	792
PATIENT: SPECIFIC		Case study 2	792
CHALLENGES TO PARAMEDIC		1. Assess	792
REASONING	750	2. Confirm	793
Chapter 43: Acute abdominal pain	751	3. Treat	793
Introduction	751	4. Evaluate	794
Anatomy	751	Case study 3	794
Pathophysiology	753	1. Assess	794
Case study 1	761	2. Confirm	795
1. Assess	761	3. Treat	796
2. Confirm	764	4. Evaluate	796
3. Treat	764	Research	796
4. Evaluate	765	Summary	796
Ongoing management	765	Chapter 45: Bleeding from the	
Hospital admission	766	gastrointestinal or urinary tract	798
Appendicitis across the lifespan	766	Introduction	798
Case study 2	766	Pathophysiology	799
1. Assess	766	Case study 1	801
2. Confirm	766	1. Assess	801
3. Treat	767	2. Confirm	802
4. Evaluate	767	3. Treat	804
Case study 3	768	4. Evaluate	804
1. Assess	768	Ongoing management	804
2. Confirm	768	GI/urinary tract haemorrhage in the field	804
3. Treat	769	Case study 2	805
4. Evaluate	769	1. Assess	805
Case study 4	770	2. Confirm	805
1. Assess	770	3. Treat	806
2. Confirm	770	4. Evaluate	806
3. Treat	771	Case study 3	807
4. Evaluate	771	1. Assess	807
Ongoing management	772	2. Confirm	808
AAA across the lifespan	772	3. Treat	809
		4. Evaluate	809

Hospital management	810	Long-term role	850
Summary	810	ESKD across the lifespan	850
<b>SECTION 15: THE PARAMEDIC APPROACH TO COMPLEX CASES: SPECIFIC CHALLENGES TO PARAMEDIC REASONING AND MANAGEMENT</b>		<b>Case study 2</b>	<b>851</b>
<b>Chapter 46: The socially isolated patient</b>	<b>812</b>	1. Assess	852
Introduction	813	2. Confirm	852
Background	814	3. Treat	852
<b>Case study 1</b>	<b>816</b>	4. Evaluate	853
1. Assess	817	<b>Case study 3</b>	<b>853</b>
2. Confirm	818	1. Assess	853
3. Treat	818	2. Confirm	854
4. Evaluate	819	3. Treat	854
Ongoing management	820	4. Evaluate	850
Hospital admission	820	Future research	854
<b>Case study 2</b>	<b>820</b>	Summary	855
1. Assess	821	<b>Chapter 49: Indigenous Australian patients</b>	<b>856</b>
2. Confirm	822	Introduction	856
3. Treat	822	Kinship	856
4. Evaluate	823	Culture	857
Summary	823	Distribution	859
<b>Chapter 47: The dying patient</b>	<b>825</b>	Epidemiological profile	860
Introduction	825	<b>Case study 1</b>	<b>861</b>
Pathophysiology	825	1. Assess	861
<b>Case study 1</b>	<b>828</b>	2. Confirm	863
1. Assess	828	3. Treat	863
2. Confirm	829	4. Evaluate	864
3. Treat	830	<b>Case study 2</b>	<b>864</b>
4. Evaluate	831	1. Assess	865
Ongoing management	832	2. Confirm	865
Hospital admission	832	3. Treat	866
Death and dying across the lifespan	833	4. Evaluate	866
<b>Case study 2</b>	<b>834</b>	<b>Case study 3</b>	<b>866</b>
1. Assess	834	1. Assess	866
2. Confirm	835	2. Confirm	867
3. Treat	836	3. Treat	868
4. Evaluate	836	4. Evaluate	868
Summary	836	Summary	869
<b>Chapter 48: The patient on out-of-hospital dialysis</b>	<b>838</b>	<b>Chapter 50: Māori patients</b>	<b>870</b>
Introduction	838	Introduction	870
Pathophysiology	838	Specific aspects of healthcare for Māori	870
Dialysis	841	Epidemiological profile	871
<b>Case study 1</b>	<b>845</b>	Delayed access to healthcare	874
1. Assess	846	Death among Māori populations	874
2. Confirm	847	<b>Case study 1</b>	<b>875</b>
3. Treat	848	1. Assess	875
4. Evaluate	850	2. Confirm	876
Ongoing management	850	3. Treat	876
Follow-up	850	4. Evaluate	877
		<b>Case study 2</b>	<b>877</b>
		1. Assess	877
		2. Confirm	877
		3. Treat	877
		<b>Case study 3</b>	<b>877</b>
		1. Assess	878
		2. Confirm	878

3. Treat	878	Case study 1	925
4. Evaluate	878	1. Assess	925
Summary	878	2. Confirm	928
		3. Treat	929
<b>SECTION 16: THE PARAMEDIC APPROACH TO THE PATIENT DISPLAYING ABNORMAL BEHAVIOUR</b>	<b>880</b>	Case study 2	934
<b>Chapter 51: The patient displaying abnormal behaviour</b>	<b>881</b>	1. Assess	935
Introduction	881	3. Treat	936
Pathophysiology	882	Hospital admission	934
Explanatory models	884	Investigations	934
Law and mental health	885	Follow-up	934
Case study 1	887	Case study 3	939
1. Assess	887	1. Assess	939
2. Confirm	893	3. Treat	941
3. Treat	894	Case study 4	942
4. Evaluate	896	1. Assess	942
Specific treatment guidelines	896	3. Treat	943
Investigations	896	Case study 5	944
Hospital admission	896	1. Assess	944
Long-term treatment and impacts	901	3. Treat	945
Mental illness across the lifespan	901	4. Evaluate	947
Case study 2	902	Further research	947
1. Assess	902	Summary	947
2. Confirm	903	<b>Chapter 54: Neonatal resuscitation</b>	<b>949</b>
3. Treat	904	Introduction	949
4. Evaluate	905	Pathophysiology	950
Case study 3	905	Identifying the newborn at risk of disorders during transition	951
1. Assess	905	Failure to breathe effectively at birth	953
2. Confirm	906	Failure to establish effective ventilation after birth	955
3. Treat	907	Preparing for the birth of a baby	955
4. Evaluate	907	Case study 1	956
Case study 4	908	1. Assess	956
1. Assess	908	2. Confirm	960
2. Confirm	908	3. Treat	961
3. Treat	909	4. Evaluate	961
4. Evaluate	910	Newborn airway management	961
Summary	910	Ongoing management	964
<b>Chapter 52: De-escalation in the pre-hospital environment</b>	<b>912</b>	Documentation	966
Introduction	912	Birth during transport	966
Theories of aggression	912	Discontinuing resuscitation	967
Management of aggression	913	Hospital admission	967
Principles of de-escalation	913	Investigations	968
De-escalation in practice	913	Follow-up	968
<b>SECTION 17: THE PARAMEDIC APPROACH TO OBSTETRIC AND NEONATAL EMERGENCIES</b>	<b>916</b>	Case study 2	968
<b>Chapter 53: Imminent birth</b>	<b>917</b>	1. Assess	969
Introduction	918	2. Confirm	970
Physiology	918	3. Treat	970
		4. Evaluate	971
		The preterm baby	969
		Long-term role	971
		Case study 3	971
		1. Assess	972
		2. Confirm	972
		3. Treat	972
		4. Evaluate	973



Case study 4	973	Principles of management	992
1. Assess	974	Summary	992
2. Confirm	974		
3. Treat	974	Chapter 57: The inflammatory response	993
4. Evaluate	975	Introduction	993
Hospital management	976	What is inflammation?	993
Future research	976	The basics of normal inflammation	993
Summary	976	Abnormal inflammation	998
		Summary	999
<b>PART 3</b>		Appendix 1: Professional role guide	1001
<b>ESSENTIAL KNOWLEDGE 979</b>		Introduction	1001
SECTION 18: ESSENTIAL CONCEPTS OF PARAMEDIC PRACTICE 980		Australasian ambulance services	1002
Chapter 55: Perfusion 981		Recruitment	1002
Introduction	981	Paramedics Australasia	1003
What is perfusion?	981	Future career paths	1003
The basics of normal perfusion	982	Alternative career paths	1003
Disturbances of perfusion	983	Other resources	1007
Assessment of perfusion	984	Appendix 2: Medications commonly encountered in community emergency health	1008
Principles of medical management of perfusion	985	Glossary	1015
Summary	986	Index	1027
Chapter 56: The autonomic response 987			
Introduction	987		
What is the autonomic nervous system?	987		
Assessment of autonomic nervous system function	991		

# Foreword

Matt Johnson, Leanne Boyd, Hugh Grantham, Kathryn Eastwood and the contributing authors are some of Australia and New Zealand's most experienced paramedics, educators, researchers and emergency physicians. So it was with great pleasure that I accepted the invitation to write this foreword for *Paramedic Principles and Practice ANZ*, a unique and valuable resource that integrates knowledge and decision making in the Australian and New Zealand context that they know and understand so well.

Paramedics are required to adapt and improve their range of clinical capabilities to provide care. This brings increased responsibility as professional clinicians to be aware of the potential impact they have on the lives of others. This impact cannot be underestimated.

The shift of paramedic education from the vocational to a university model has resulted in clinicians who enter the workforce with a complex understanding of anatomy, physiology, pathology and pharmacology. There is little doubt that this science has been an important step in the development of the paramedic profession. However, new graduate paramedics now have much less clinical exposure where they can learn the art of being a paramedic. I am impressed with the way this text lays out the pathway for graduates to develop and grow to be expert clinicians by bringing together the art and science of paramedicine. The inclusion of real-life stories reinforces this message and brings to life important theoretical models related to developing as an expert clinician and lifelong learner.

This text goes beyond the technical aspects of emergency care: it drives and reinforces the importance of professional attitudes, behaviours,

clinical competence, teamwork, communication skills and the humanitarian approach required of paramedics. It is a refreshing approach to the complex challenges paramedics face in the context of an ageing population, high instances of chronic health problems, a health system that offers limited access to community-based clinicians and limited technological assistance for paramedic decision making. This book will be a valuable tool for those wanting to provide high-quality, patient-focused care in this challenging healthcare environment.

Healthcare starts at the patient, not at the emergency department or at a hospital or clinic door. In this context it is notable that decisions and clinical interventions performed by paramedics often keep patients alive until they can receive more definitive care.

Paramedic assessments, decisions and interventions have the capacity to keep patients out of the hospital system entirely, reduce morbidity and reduce the length of hospital stay, all of which have the potential to reduce the social and economic burden on the health system.

I recommend this text to you as a resource that will assist you to contribute confidently to the care of your community and to continue to develop your professional practice and career.

*Excellence is an art won by training and habitation.*

*We do not act rightly because we have virtue or excellence but rather we have those because we have acted rightly, we are what we repeatedly do.*

*Excellence is not an act but a habit.*

Aristotle (384–322 BC)

Adjunct Professor Ian Patrick ASM, FPA, LMPA

# Preface

*Medicine is a science of uncertainty and an art of probability. One of the chief reasons for this uncertainty is the increasing variability in the manifestations of any one disease.*

Sir William Osler (1849–1919)

You cannot become a good paramedic by reading a book, regardless of how good the book or how great your memory. Certainly, every case you attend as a paramedic will require you to call upon a detailed knowledge of anatomy, physiology and pharmacology, but to practise effectively in the field of emergency medicine you also need to be able to interpret people and situations. While exams require you to recall facts with certainty, clinical practice is too often lacking in both facts and certainty.

To be a safe paramedic you need to elicit accurate information from patients who will differ from you in gender, generation, social situation and health. You then have to determine what (if any) of that information is relevant and finally draw together all of these knowledge sets, skills and attitudes to determine a diagnosis.

The process of clinical reasoning is probably the most difficult for students of any medical discipline to learn. Anatomy, physiology and pharmacology will come easily to those blessed with a good memory but will also eventually sink into the minds of the rest of us. Similarly, guidelines and clinical skills can be learned by practice. But how do you take all of this knowledge and use it when you are confronted with a patient? Traditional teaching and texts stretching back 100 years suggest you must first learn the basic biomedical sciences before you engage in solving clinical problems. While the editors of this text do not disagree entirely, it does raise the question: how do you manage a condition if you have not been taught the specifics of the disease that caused it?

An alternative method is to apply the clinical reasoning approach to conditions as you learn them. This text does not substitute for other texts which, in far more detail, describe the anatomy, pathophysiology and pharmacology you will need to pass your exams. What the editors hope to offer is a text that allows you to see the links between the pathophysiology of a disease, how this creates the signs and symptoms perceived by the patient and how these need to be managed in the out-of-hospital environment.

Clinical reasoning is a real-time, living mystery. Traditional teaching methods offer you the clues that allow you to solve the clinical puzzle. But in real life, paramedics have to extract and sort the clues by importance before they must decide on an answer. To help you to develop this skill, this book is structured in three parts. The chapters in Part 1 articulate the principles that support good paramedic practice: the ability to communicate effectively, gather essential clinical information in difficult environments and use this information to make safe and effective clinical decisions. The chapters in Part 2 present the various conditions that novice paramedics can expect to see as they practise, with each chapter revealing the clinical reasoning process and describing the principles of management for a particular condition. To do this we use a series of case studies, stepping you through each case scenario to link the clues and, importantly, reveal the process of reaching a safe and effective management plan. Finally, the chapters in Part 3 outline three essential concepts that underpin paramedic practice and are common to a wide range of diseases and injuries: perfusion, the autonomic nervous system and inflammation. The importance of understanding how these concepts integrate into the disease process cannot be overemphasised.

# The key to improving your clinical practice

The ability to reach safe and accurate clinical decisions is not solely influenced by the paramedic's knowledge of disease and dependence on guidelines. The expectations of the patient and their family, the patient's language, age, gender, experience and fatigue, and the need to manage scarce resources all impact on the paramedic's ability to exercise effective clinical judgement and deal with the uncertainty that surrounds any diagnosis. This text therefore aims not simply to link pathology with symptoms, but also to contextualise paramedic practice to reveal the unique strategies that experienced paramedics use to practise effectively in the field. Under time and resource pressures, the best paramedics are often unaware of the decision-making process they use to devise treatment plans: this book is designed to reveal this process and provide students with a guide to making safe and effective clinical decisions.

To do this, each chapter in Part 2 presents several case studies based on a genuine ambulance dispatch, because in the real world of paramedic practice the problem-solving process starts with the dispatch: differential diagnoses and treatment plans spring to mind the moment a job is received. The initial case study in each chapter describes the typical presentation for a particular condition and the signs

and symptoms that should not be missed. It identifies a particular pathology and how this links to the patient's signs and symptoms, and outlines how the condition commonly presents, the clinical decision-making challenges often associated with it and how it can be managed. As each chapter progresses, the presentation becomes less 'classic' and the chapter describes how to reach a clinical decision when faced with increasing uncertainty. The subsequent case studies in each chapter thus present less-typical examples, enabling novice practitioners to examine the decision-making processes of more experienced clinicians and to explore how safe and effective clinical decisions can be made.

Understanding how you make clinical decisions is the key to improving your skill, and learning why clinicians make errors in collecting or processing information allows you to identify these behaviours in yourself and correct them. Even for experienced clinicians, there is value in understanding the process: when experts are faced with a condition they haven't encountered before, they may switch to the form of clinical reasoning more often used by novices. This meta-cognition (analysing how you think) is what separates expert emergency clinicians from those with simply a good memory.

## Key features

### CASE STUDY 1

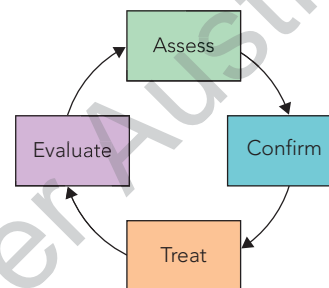
#### Case 10692, 0006 hrs.

**Dispatch details:** Three males have been assaulted outside a nightclub. One has facial injuries. Police on the scene state that another patient may have been stabbed.

**Initial presentation:** The paramedics find one patient standing outside the nightclub holding a bloodied towel to his face and another two patients sitting in the gutter talking to the police. The police direct the male with facial injuries towards the paramedics. He is distressed but not aggressive and removes the towel to reveal a lacerated lip and a missing tooth. One of the two men sitting in the gutter has a torn shirt but no obvious injuries or blood. Both men are talking calmly to the police. The men who assaulted the group have left the scene.

**Case study** Each case study is based on a genuine ambulance dispatch and reflects the years of experience of the various chapter authors. The introduction section to each case outlines the dispatch number and call-out time, the dispatch details as noted by the call-taker and the patient's initial presentation when the paramedics arrive on the scene.

**Clinical reasoning steps** For every case study we take you through the four-step clinical reasoning process of **assess**, **confirm**, **treat** and **evaluate**. This process is critical to improving your clinical decision making; although the process is rarely visible in practice, nearly every clinician practising medicine uses this model.



- 1 **Assess** Assessment of patients in the pre-hospital setting is limited in both time and equipment. An accurate history and a structured approach are the two most important elements. Each case study identifies the most pertinent signs and symptoms associated with a particular condition.
- 2 **Confirm** In addition to linking the pathophysiology of a condition with the symptoms, each case study details the various differential diagnoses that should be considered when you are presented with a particular case history. This reflects the way experienced clinicians think: exploring a range of potential causes for a patient's condition and systematically eliminating them until the best management plan is reached.
- 3 **Treat** Each case study outlines the principles of managing the condition with special consideration for the pre-hospital environment. The treatments are not based around a particular skill set, but start with the most basic elements and extend through to an intensive care level.
- 4 **Evaluate** The evaluation phase is important as the patient's response to treatment can indicate the accuracy of the initial diagnosis. Not all treatments available to paramedics demonstrate an effect in the timeframes usually associated with emergency care, and in some cases you should expect to see a realistically small or absent response to treatment. In a few cases (as in real practice) the treatment will inevitably be futile and the patient will not survive.

**Ongoing management** We conclude the case studies with an outline of what happens once the patient arrives at hospital, because understanding where pre- and in-hospital treatments align and differ can assist in determining how aggressive pre-hospital management should be and where transport should be considered as treatment.



## Asthma

By Paul Olivera and Matt Johnson

### OVERVIEW

- Characterised by wheezing, breathlessness, chest tightness and coughing, the spectrum of patient presentations for asthma ranges from mild symptoms to cardiac arrest.
- There were 416 deaths from asthma in Australia in 2010 (National Asthma Council, Australia, 2013).
- In severe forms of asthma, airflow restriction can compromise alveolar ventilation and respiration, leading to hypoxia and hypercapnia.
- The increased resistance to airflow can trap gas in the lungs and, with repeated efforts to ventilate, patients can raise intrathoracic pressures so high that venous return to the heart is restricted. This can result in loss of blood pressure and loss of consciousness.
- Once considered to be an acute disease there is now acceptance that chronic changes in the airways contribute to disease severity.
- In the acute setting the condition is diagnosed clinically and without specific tests. Diagnosis relies heavily on an accurate history and respiratory assessment.
- With more than 80% of people with asthma suffering from allergies and the condition having numerous common and individual triggers, there are many similarities between asthma and the allergy/anaphylaxis pathophysiology pathway.

### CONCEPTS USED IN THIS CHAPTER

- The paramedic's clinical approach: Section 2
- Perfusion: Chapter 55
- The autonomic response: Chapter 56
- The inflammatory response: Chapter 57

### Introduction

Asthma is a chronic inflammatory disorder of the small airways characterised by episodes of acute exacerbation. The disorder causes airflow restriction to the alveoli as a result of concurrent airway constriction and obstruction. Once considered to be a simple disease, it is now understood to be a complex and multifactorial collection of conditions that limit airflow through both acute and chronic airway changes (Myers & Tomasio, 2011; Sactta & Turato, 2001).

### Pathophysiology

The ability to conduct air quickly from the mouth and nose to the alveoli of the lungs is essential for survival, but it also provides a portal through which viruses, bacteria, fungi and other contaminants can enter the body. This combination of necessity and vulnerability has determined the structure and anatomy of the human airway.

Subject to large pressure changes and high rates of flow, the upper airway is reinforced by cartilage and lined with mucus-secreting cells and cilia to help trap and remove tiny foreign bodies. The small airways (bronchioles) are subject to much slower airflow and are not supported by cartilage.

They are made up of rings of smooth muscle and elastic tissue lined with smooth epithelial cells and mucus-secreting cells. Between the inner and outer layers of the small airways are numerous immune cells such as mast cells, eosinophils and neutrophils (see Fig 17.1). When the immune cells detect a pathogen, they initiate an inflammatory response (see Ch 57). The release of inflammatory mediators (histamine, eosinophilic and neutrophilic chemotactic factors, leukotrienes, prostaglandin and cytokines) from the immune cells causes:

- the smooth muscle lining the airway to contract (bronchoconstriction)
- the goblet cells to secrete more mucus into the lumen (mucus plugging)
- the airway walls themselves to become oedematous (due to vascular congestion and increased capillary permeability; Curtis & Ramsden, 2011).

As a result, the airway lumen becomes smaller and resistive to airflow (see Fig 17.2).

All of these responses are aimed at trapping the pathogen, preventing its progression deeper into the airway, and reflect a normal immune reaction (Murphy & O'Byrne, 2010). In the asthmatic patient, however, the complex interaction between

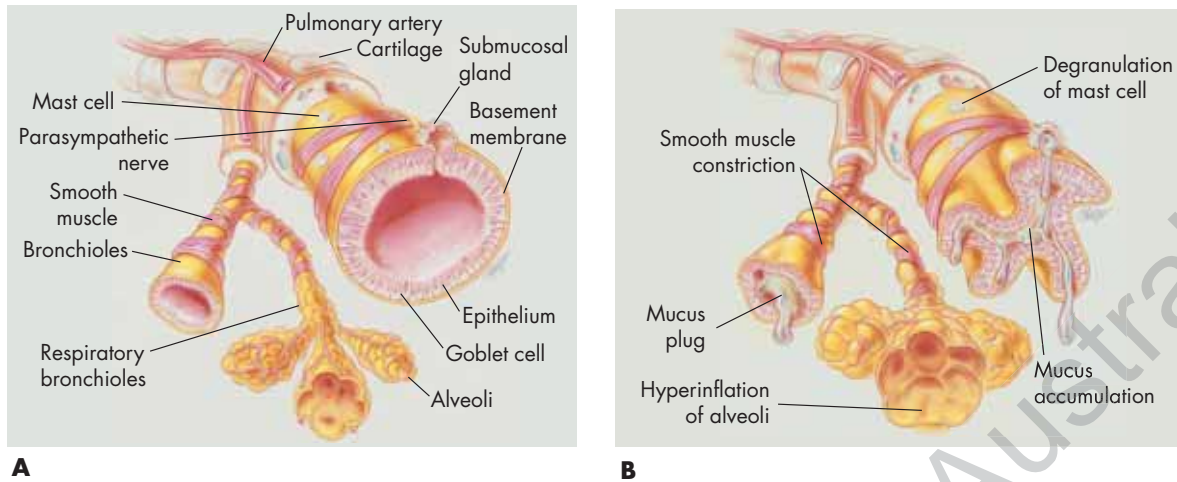


Figure 17.1

**A** Normal lung with clear airways. **B** Thick mucus, mucosal oedema and smooth muscle spasm causing obstruction of small airways occurs in asthma, breathing becomes laboured and expiration is difficult due to the airway restrictions.

Source: Des Jardins & Burton (2006).

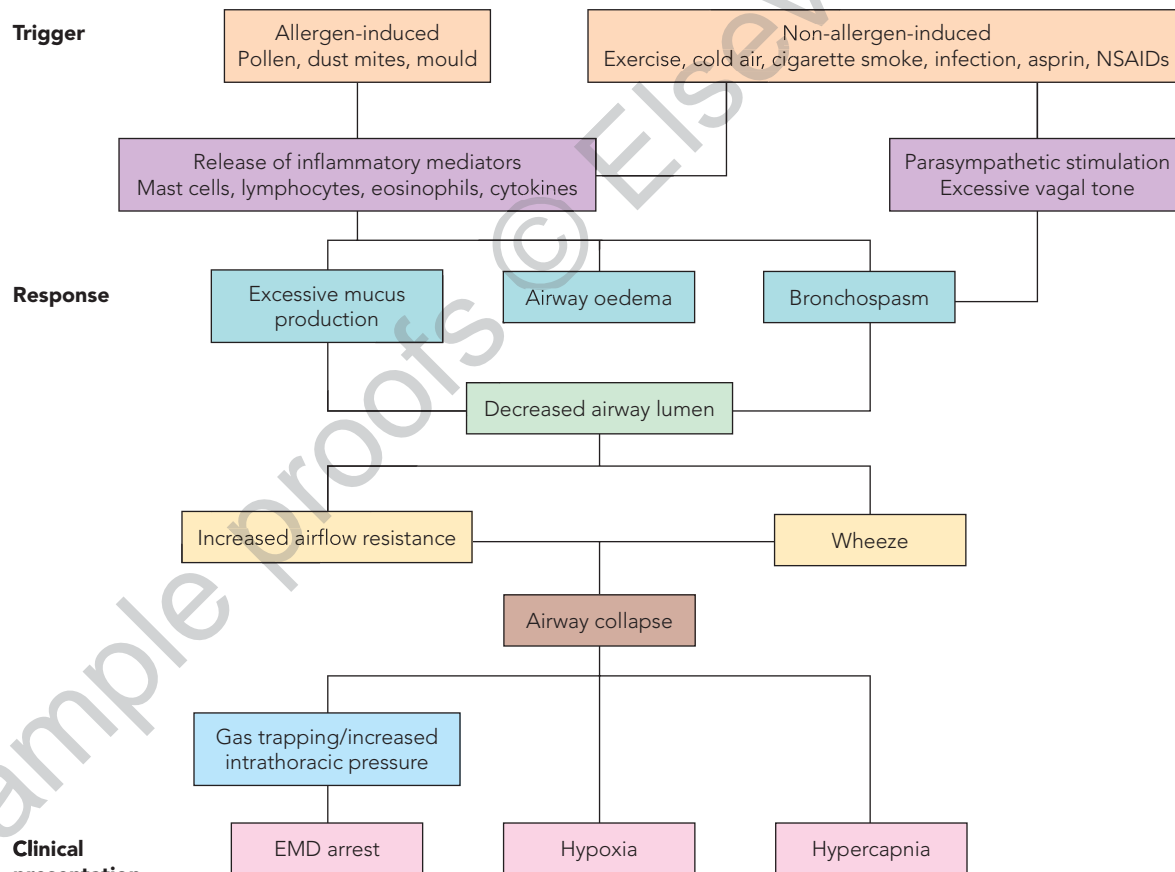


Figure 17.2

#### Asthma pathway.

Asthma is usually the result of the development of IgE-mediated response to common allergens. Combined with a hyper-reactive airway, the inflammatory mediators trigger the classic triad of increased mucus production, airway swelling and bronchoconstriction. This causes increased airflow resistance and ultimately the inability to ventilate. The non-allergen triggered pathway also triggers an abnormal inflammatory response and may include an abnormal autonomic response.

the inflammatory mediators is overly sensitive and produces a response far in excess of a normal reaction. This leads to an excessive degree of airway restriction across a wide area of the lung (Murphy & O'Byrne, 2010; see Box 17.1). Although small airway inflammation is not unique to asthma (it occurs locally in most chest infections and in other respiratory diseases), asthma is characterised by a hyperresponsive inflammatory reaction that can occur in areas of the lung not directly affected by the pathogen (Rodriguez-Roisin, 1997).

For the patient, the constriction of the small airways increases resistance to airflow and makes the effort of breathing more difficult. To overcome this increased resistance patients recruit muscles other than the diaphragm to move air in and out of the alveoli. In mild asthma this sense of 'increased work of breathing' or 'chest tightness' can be distressing to patients but, with effort, they are usually able to maintain normal alveolar ventilation and may even hyperventilate in response to the anxiety of a possible impending asthma attack. As such, the levels of oxygen and carbon dioxide ( $\text{CO}_2$ ) in the blood remain at near-normal levels (Rodriguez-Roisin, 1997). If the degree of airway obstruction is severe, however, and the subsequent increase in airflow restriction exceeds the ability of the muscles of ventilation to overcome the pressures generated, the amount of air reaching the alveoli eventually falls and the ability to exchange oxygen and  $\text{CO}_2$  is compromised.

Clinically, this airway narrowing is linked to wheezes heard with or without a stethoscope. The exact mechanism by which wheezes are caused remains unclear (Spence et al., 1996), but they are commonly described as 'musical' and 'continuous'. In reality the volume and tone of wheezes are actually quite variable and they may last for as little 250 ms (Pasterkamp, Kraman & Wodicka, 1997). Although vibration of airway secretions may contribute to wheezes, it is widely held that they are caused by vibrations of the airways caused by turbulent airflow (Gavriely et al., 1989; Pasterkamp, Kraman & Wodicka, 1997). Predominantly heard on expiration (as the airways get smaller and the walls come into closer proximity as a result of the thoracic muscles contracting the chest wall), the wheeze's pitch, volume, duration and location in the respiratory cycle have variously been described as indicators of the severity of airway narrowing. However, given the dynamic nature and unpredictability of asthma presentations, these factors are probably not predictive in the emergency environment.

### BOX 17.1 Asthma triggers

With asthma rates on the rise in western nations, there has been considerable research into the reason why this immune-based condition is becoming more common. The body's normal immune response protects against antigens through antibody recognition of the antigen, followed by the release of a cascade of inflammatory mediators that are designed to isolate and destroy the antigen. Allergic reactions are excessive reactions by these antibodies, and diseases such as asthma, anaphylaxis and hay fever share the common pathway of a supposedly protective reaction that has overreacted and become harmful.

The tendency to produce the particular type of antibodies associated with allergies has some genetic component but also appears to be related to the amount of exposure.

Many of the triggers that cause asthma are typical antigens associated with allergies: house mites, animal hair and pollen. Drugs that interfere with the inflammatory response (such as aspirin, ibuprofen and ACE inhibitors) can also upset the delicate inflammatory pathway, but interestingly non-antigen-based triggers such as cold air, exercise, stress and smoke can also trigger an inflammatory response in the lungs. Although they may differ in a chemical sense, the clinical manifestations of both the immunological and the non-immunological pathways result in the same presentation.

The limited predictive nature of wheezes is reinforced by numerous studies that have shown that while airflow is always limited when a wheeze is present, that flow may also be limited without wheeze (Gavriely & Grotberg, 1988; Gavriely et al., 1989; Pasterkamp et al., 1997). Theoretical models of airway vibration suggest that airway wall thickness and longitudinal tension affect the nature of a wheeze and these factors change during each ventilation (Amatoury et al., 2010). As a result, mild-to-moderate asthma can present with a combination of slight increased work of breathing with or without a persistent cough, and either with or without wheezes. The progression from mild to severe (and potentially fatal) asthma involves an increase of airflow restriction that induces both chemical and mechanical changes to ventilation and perfusion.

### Chemical alterations due to severe airway constriction

Human alveoli are often ventilated by more than one bronchiole. The canals of Lambert connect

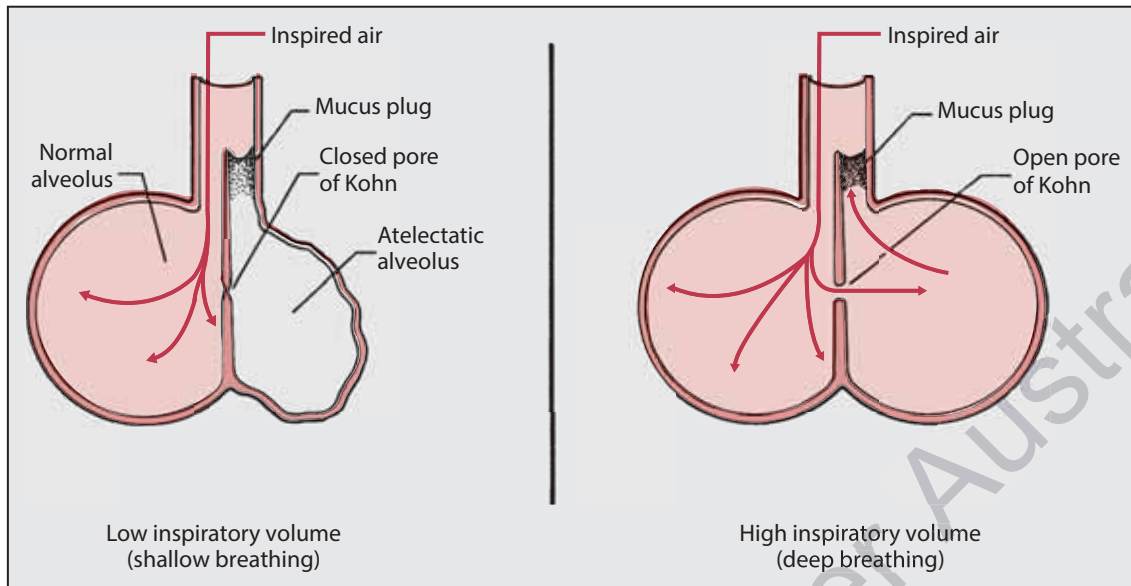


Figure 17.3

The pores of Kohn connect alveoli and may allow ventilation when the bronchiole is obstructed. In the early stages of an acute asthma attack this may allow asthmatics to maintain normal blood chemistry despite significant changes occurring in the small airways.

Source: McCance & Huether (2014).

bronchioles with adjacent alveoli while the pores of Kohn connect neighbouring alveoli to each other (Wyka, Mathews & Clark, 2011; see Fig 17.3). These passages of collateral ventilation allow gas flow even when some bronchioles become completely blocked. But eventually the restriction of air entering the lungs will impact on gas exchange across the alveolar membrane. The resultant hypoxia elicits a sympathetic response (see Ch 56) that causes an initial rise in heart rate and blood pressure. The respiratory acidosis that results from the inability to remove  $\text{CO}_2$  increases the sense of respiratory distress and, combined with hypoxia, will ultimately lead to a decreased conscious state if not corrected. Typically in asthma, the combination of hypoxia and hypercapnia leads to a period of confusion or catatonia prior to a complete loss of consciousness, but the duration of the phase is unpredictable as it depends on the degree of respiratory compromise and the speed with which it developed.

If the hypoxia becomes entrenched, eventually the heart rate will start to decrease, as the myocardium cannot maintain normal function. Slowing of the heart rate combined with a falling level of consciousness should be considered late and extremely poor signs. The bradycardia can lead to a subsequent fall in blood pressure but hypotension in asthma is more often due to a mechanical cause

and can be sudden, catastrophic and occur with little change in heart rate.

#### Mechanical alterations due to airway constriction

Gentle and unconscious contraction of the diaphragm is usually sufficient to draw air into the lungs. To inflate the lungs beyond the normal tidal volume the diaphragm can contract further and the muscles that attach to the ribs can be used to further increase the intrathoracic volume. Because the ribs in adults slope downwards from their point of articulation on the spine, pulling the ribs upwards increases the distance from the front of the chest wall to the posterior wall. This is a strong and effective way of expanding the intrathoracic volume, creating a negative intrathoracic pressure and drawing air into the lungs. The biomechanics of both diaphragmatic contraction and respiratory accessory muscle use favour ventilation near the normal tidal volume range and the effectiveness of both of these mechanisms in thorax expansion decreases as they reach the limits of expansion (Stather & Stewart, 2005; see Fig 17.4).

The lungs themselves also affect the efficiency of ventilation. The force required to inflate the lungs increases exponentially as the lungs inflate and the tissues are stretched. The net effect of both the mechanical disadvantage of the



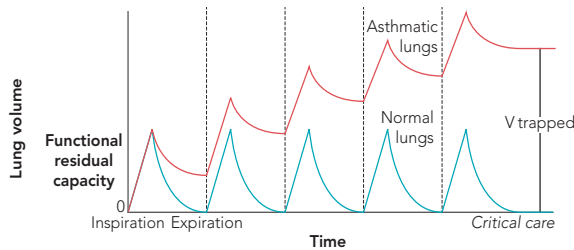


Figure 17.4

**Mechanism of dynamic hyperinflation in the setting of severe airflow obstruction.**

With narrowing of the small airways, the normal elasticity of the lungs is not sufficient to expel inhaled air and the patient is forced to use muscular effort to force the inspired gas out of their lungs. Gas trapping occurs because the inspiratory reflex occurs before all the air from the previous inspiration has been expired.

Source: Stather & Stewart (2005).

muscles of respiration and the elasticity of the lungs is that it takes far more energy to ventilate at or near the top of the vital capacity than near the middle of the ventilation range (Stather & Stewart, 2005).

This is especially important in asthma as the airway constriction creates a unique effect whereby the mechanical forces of inhalation are usually able to overcome the increased airway resistance, but the narrowed airways then collapse on exhalation, trapping air between the alveoli and the small airways. This occurs because as the chest wall muscles relax, the chest wall drops, creating an increased intrathoracic pressure. This pressure is exerted upon the small airways, which narrow due to the lack of cartilaginous support. The effect of this is that the air in the terminal airways is now partially trapped, gradually leading to hyperinflation of the lungs. This is known as dynamic hyperinflation or 'gas trapping'.

In the early stages the effects of gas trapping on blood chemistry (hypoxia and hypercapnia) are often negligible as the reduced volume inhalations (because of gas already being present) are often sufficient to maintain arterial oxygen levels. Unfortunately, however, once the patient reaches their vital capacity they cannot inhale any further and hypoxia will quickly develop. Another consequence of gas trapping is that the hyperinflated lungs and associated increased intrathoracic pressure can occlude the vessels (such as the inferior and superior vena cava) returning venous blood back to heart. This compromise of central venous pressure (CVP) will lead to a fall in cardiac output and blood pressure.

The exact mechanism of gas trapping is not entirely clear and probably involves not only airway narrowing, but also dyssynchrony of the muscles of ventilation as they fatigue or near their limits of mechanical advantage (Stanley & Tunnicliffe, 2008; Stather & Stewart, 2005). Predicting the progression of gas trapping and the degree to which it will affect blood pressure and blood chemistry is difficult to determine in the emergency setting, but it is a vital concept to understand if paramedics are to effectively manage unconscious asthmatic patients.

### Airway remodelling

The past two decades have seen a dramatic change in the understanding of asthma pathophysiology, in particular a shift from the view of the disease as being acute and mostly reversible between outbreaks to being a disease where significant changes can occur to the airway between acute episodes. This chronic remodelling of the smaller airways consists of two major components: the infiltration of the airway wall by inflammatory cells; and the structural thickening of the airway wall itself (Saetta & Turato, 2001).

### Inflammatory cell infiltration

The dysfunctional interaction that occurs in asthma between mast cells, eosinophils, neutrophils and lymphocytes is still not completely understood, but histological examinations of chronic asthmatic airways usually reveal excessive populations of eosinophils (Saetta & Turato, 2001). The abundance of eosinophils almost certainly contributes to hyper-reactive inflammatory response, but more recently a small group of asthmatics have been found to also have excessive populations of neutrophils within their airway walls. This group of asthmatics typically suffers from severe symptoms but it remains unclear if the neutrophils are the cause of symptom severity, are a response to the medications used to treat severe asthma (Cox, 1995) or are unrelated to the disease severity (Saetta & Turato, 2001). As a result of these findings several researchers have described the two pathophysiologies as two separate phenotypes of asthma.

### Structural changes

While the exact contribution that the increased populations of eosinophils and neutrophils plays in the chronic development of the disease remains unclear, they are just one of the chronic changes to the airway wall that occur in asthma. Increased amounts of smooth muscle, deposits of collagen, hypertrophy of mucus-secreting cells, vascular congestion and thickening of the basement



membrane that attaches the epithelial tissue to the airway are all evident on examination (Saetta & Turato, 2001). By adding to the bulk of the airway wall, all these factors place pressure on the lumen and reduce the diameter of the airway, but some also add to the degree of constriction

(bronchospasm) and obstruction (mucus production) when an acute episode occurs. The thickening of the basement membrane may be of particular importance in asthma as it is not shared by other chronic airways diseases such as COPD (Saetta & Turato, 2001).

## CS CASE STUDY 1

### Case 10364, 1030 hrs.

**Dispatch details:** A 23-year-old female university student waiting to enter an exam room has become extremely short of breath.

**Initial presentation:** The patient is sitting upright on a chair in an office to the side of the exam hall. She has one arm by her side and is holding the flat palm of the other on her chest. She is conscious, alert and breathing rapidly with a clear rise and fall of the chest. She appears mildly distressed. She says that she thinks she is suffering from asthma and has used her friend's Ventolin inhaler. The patient states she had asthma as a child but only uses relievers occasionally now.

## 1 ASSESS

### Patient history

Patients presenting with disturbances of conscious state, breathing or perfusion are often in need of rapid intervention and paramedics must recognise that they may not always have the luxury of obtaining a full clinical history. As described in Chapter 4, focusing the history on differentiating between the causes of sudden respiratory distress can allow for a quicker and more succinct process. Applying an understanding of the epidemiology and pathology of a disease will assist you in determining which aspects of the history are important and which are irrelevant.

Asthma is an episodic disease that is usually diagnosed before children commence primary school. Patients who present with shortness of breath caused by asthma often have a history of asthma. If the patient has a past history of asthma, this is a strong indication that their shortness of breath may be caused by an inflammatory response leading to narrowing of the airways. In the opening case study the patient says she had severe asthma as a child, but only uses a reliever occasionally now.

The involvement of both the inflammatory response and the immune response in asthma means that up to a third of acute asthma episodes are preceded by a chest infection (Sutherland & Martin, 2007; Teichtahl, Buckmaster & Pertnikovs, 1997) and some sufferers report that their asthma symptoms occur only when they are suffering a chest infection. More worrying is the disproportionate rate of fatal asthma episodes in young adults who had supposedly 'grown out' of their childhood asthma. Pathologically, these patients may have developed chronically inflamed airways that they have managed symptomatically but, when exposed to a particular allergen, the airways are

## HISTORY

### Ask!

- Have you had any previous episodes of asthma?
- Have you had any previous episodes of shortness of breath?
- Have you had any chest colds/infections recently?
- How has your breathing been at night recently?
- Is this as bad as your asthma ever gets?
- Are the symptoms getting better or worse?
- Do you know what triggers your asthma?

‘primed’ to react aggressively. This may be compounded by the patient’s lack of early recognition of the symptoms and the lack of a reliever medication.

Identifying a past history of asthma early in the assessment is clearly important and many paramedics also ask if patients have been previously admitted to hospital with their asthma. While this can provide a gauge of how severe their asthma has been in the past, it does not take into account the impact of chronic airway changes and may not assist in determining whether the current presentation is asthma or another form of respiratory distress. A more reliable predictor of gradually worsening asthma is a nocturnal pattern of dyspnoea (Saetta & Turato, 2001; see Box 17.2).

When the patient in the case study is questioned further, she says she has needed to use her reliever at night for about a week but hasn’t had a chest infection.

#### Peak expiratory flow measurement

The role of out-of-hospital peak expiratory flow (PEF) measurement is controversial. A peak flow meter is used to detect and measure a person’s variation in best PEF, in order to assess variability of airflow limitation (National Asthma Council Australia, 2006). Measurement of PEF:

- is effort-dependent
- varies considerably between instruments.

Isolated readings taken with a meter other than the person’s own must be interpreted with caution because there is a wide normal range. Despite its limitations, monitoring of PEF at home or work is useful when:

- symptoms are intermittent
- symptoms are related to occupational triggers
- the diagnosis is uncertain
- monitoring treatment response (National Asthma Council Australia, 2006).

### BOX 17.2 Asthma classifications

#### Intermittent asthma

Untreated asthma is classified as *intermittent* if all of the following apply:

- Daytime asthma symptoms occur less than once per week.
- Night-time asthma symptoms occur less than twice per month.
- Exacerbations are infrequent and brief.
- FEV<sub>1</sub> (forced expiratory volume in 1 second) is at least 80% predicted and varies by less than 20%.

#### Mild persistent asthma

Untreated asthma is classified as *mild persistent* if one or more of the following applies (and more severe signs and symptoms are not present):

- Daytime asthma symptoms occur more than once per week but not every day.
- Night-time asthma symptoms occur more than twice per month, but not every week.
- Exacerbations occur occasionally and may affect activity or sleep.
- FEV<sub>1</sub> is at least 80% predicted and varies by 20–30%.

#### Moderate persistent asthma

Untreated asthma is classified as *moderate persistent* if one or more of the following applies (and more severe signs and symptoms are not present):

- Daytime asthma symptoms occur every day, but do not generally restrict physical activity.
- Night-time asthma symptoms occur at least once per week.
- Exacerbations occur occasionally and may affect activity or sleep.
- FEV<sub>1</sub> is 60–80% predicted and varies by more than 30%.

#### Severe persistent asthma

Untreated asthma is classified as *severe persistent* if one or more of the following applies:

- Daytime asthma symptoms occur every day and restrict physical activity.
- Night-time asthma symptoms occur every day.
- Exacerbations are frequent.
- FEV<sub>1</sub> is 60% predicted or less, and varies by more than 30%.

Source: National Asthma Council, Australia (2006).

### EtCO<sub>2</sub> measurement

End tidal carbon dioxide (EtCO<sub>2</sub>) monitoring is the non-invasive measurement of exhaled CO<sub>2</sub>. Donald and Paterson (2006) reviewed the clinical indication for its use in the pre-hospital environment and supported its use for asthma and advanced airway management.

### Self-management

Assessing the patient's strategies and success in managing their shortness of breath will provide valuable information. Short-acting adrenergic drugs such as Ventolin are generally effective in managing acute bronchospasm. If the patient has self-medicated and improved it is a strong indication that inflammatory-driven airway narrowing is involved in the presentation.

### Airway

The patient is conscious and talking and has not suffered any trauma. In terms of the primary survey, the airway is patent.

### Breathing

A full respiratory status assessment is essential for any patient showing signs of respiratory distress. Combined with the history, what you find during this assessment will determine your initial treatment. A common paramedic checklist for assessing respiratory status is **PASSRESPS**:

- **Position:** Sitting upright, one arm by their side. In order to maximise the use of accessory respiratory muscles patients often extend their arms forwards of their body and anchor their palms on a table, wall or flexed knees. Known as the tripod position it indicates severe airway constriction. This patient's arms are relaxed. While this doesn't exclude accessory muscle use, it shows the airways pressures are not (currently) excessively high.
- **Appearance:** The patient appears mildly anxious. Understandable given the circumstances but she is not highly distressed or catatonic: indicators of both hypoxia and hypercapnia.
- **Speech:** Speaking requires exhalation: the inability to speak in full sentences indicates some alteration in exhalation. This patient is speaking freely.
- **Sounds:** Auscultating the chest of this patient reveals mild expiratory wheezes across both lungs from the mid-zones to the bases. The widespread and equilateral nature of the wheeze suggests that the airway narrowing is not due to an infection or aspiration, which would be more localised. Remember, wheezes are generated by airflow: if the patient is poorly ventilating, wheezes will not occur. You need to reconcile the volume and degree of wheezes with the amount of air the patient is (or isn't) moving with each ventilatory effort. The airways get smaller (in diameter and length) as patients expire, so wheezes are more likely to occur on expiration. Inspiratory wheezes should be considered a serious sign when they occur in combination with expiratory wheezes.
- **Rate:** A respiratory rate above 20 is high, especially so if the expiratory phase is prolonged by gas trapping. A high respiratory rate alone can have a number of causes so it is not necessarily definitive of either the cause or the severity.
- **Rhythm:** The normal time ratio between inspiration and expiration (IE ratio) is 1:2 but airway swelling, constriction and obstruction will impact most severely on the ability to expire. Asthma is therefore characterised by a prolonged expiratory phase. Watching this patient breathe for a couple of cycles reveals a clearly prolonged expiratory phase.
- **Effort:** The use of the scalene muscles on inspiration and, particularly, the abdominal muscles on expiration are strong indicators of bronchospasm causing raised airway resistance. This patient is using both to assist her breathing.

### PRACTICE TIP

Paramedics face a real challenge when assessing a severely breathless patient who cannot answer questions if no relative or bystander is present. Limiting your questions as much as possible and asking questions requiring a yes/no response can help. Writing 'yes' and 'no' on a sheet of paper and asking the patient to point to the relevant response is another useful technique.

### RESPIRATORY STATUS

#### Look for!

- Increased work of breathing
- Ability to speak full sentences
- Prolonged expiratory phase

### CARDIOVASCULAR STATUS

#### Look for!

- Tachycardia: early
- Bradycardia: late!
- Hypotension: late!
- Altered conscious state: late!

- **Pulse:** The patient is tachycardic but the trend is as important as the number. Expect the heart rate to rise as severity increases but be aware that a reduction in heart rate with a concurrent deterioration in the patient's conscious state suggests imminent respiratory arrest. Commencing treatment with  $\beta_2$  agonists does not usually increase the heart rate of patients who are tachycardic prior to administration but it will increase the rate of less symptomatic patients.
- **Skin:** Because asthmatics are generally able to maintain a reasonable tidal volume (until the effects of gas trapping resist further inspiration), cyanosis is not common in the conscious asthmatic and should be considered a late sign. The increased effort of breathing and underlying hypercapnia often lead to flushed, warm and sweaty skin as opposed to the cool, pale and clammy skin that would be expected of a patient with a strong sympathetic drive. The skin state is not a strong diagnostic tool for asthma.

### Cardiovascular

The risk of perfusion issues associated with asthma generally rises as the duration of symptoms extends and the severity level increases. While the patient is able to keep inspiratory and expiratory volumes close to equal, the likelihood of gas trapping causing a catastrophic drop in blood pressure is unlikely. Similarly, a tachycardic response to the combination of anxiety, increased work of breathing and hypoxia is to be expected. Bradycardia is a late sign in asthma, rarely occurs in isolation and is usually associated with an altered conscious state.

### CNS

As the ability to ventilate is compromised, hypoxia and hypercapnia will occur. Initially these tend to present as anxiety and distress but, depending on the degree and speed of the ventilation failure, they are often followed by a period of confusion, aggression and then catatonia before a complete loss of consciousness occurs. This progression is typical of respiratory failure and, apart from acute hypoxia, may be associated with high levels of  $\text{CO}_2$  causing cerebral vasodilation and increased intracranial pressure.

### Initial assessment summary

Problem	Acute shortness of breath
Conscious state	Alert
Position	Sitting upright
Heart rate	Radial pulse 125
Blood pressure	110/70 mmHg
Skin appearance	Flushed, warm
Speech pattern	Able to speak in sentences
Respiratory rate	24 BPM
Respiratory rhythm	Slightly prolonged respiratory phase
Chest auscultation	Mild expiratory wheezes, L = R
Pulse oximetry $\text{SpO}_2$	98% on room air
Temperature	36.8°C
Capillary refill (seconds)	4
Pain score	0/10
Motor/sensory function	Normal
History	Asthma Became short of breath while waiting to do an exam

**D:** There is no danger to the ambulance crew or patient.

**A:** The patient is conscious with no airway obstruction.

**B:** Respiratory rate is elevated and the work of breathing is increased. There is an expiratory wheeze in both lungs that is equal left to right. Tidal volume is normal.

**C:** Heart rate is elevated; blood pressure is within normal limits.

The patient is presenting with dyspnoea at rest and has a history of hyper-reactive airways disease. Although the circumstances are consistent with anxiety-induced hyperventilation, the presence of the wheeze combined with the increased work of breathing are consistent with acute asthma.

## 2

## CONFIRM

The essential part of the clinical reasoning process is to seek to confirm your initial hypothesis by finding clinical signs that should occur with your provisional diagnosis. You should also seek to challenge your diagnosis by exploring findings that do not fit your hypothesis: don't just ignore them because they don't fit.

Paramedics are forced to diagnose asthma based on its clinical presentation: there are no definitive in-field tests. As such it is usually important to exclude as many other possible causes before commencing treatment. However, the presence of wheezes with respiratory distress, combined with the relative safety of the first-line treatment, will see many paramedics commence treating for asthma while they exclude other causes.

What else could it be?

### Anaphylaxis

Asthma and anaphylaxis share similar hyperreactive inflammatory responses and initially it can be very difficult to make a definitive diagnosis between the two. Anaphylaxis occurs across multiple systems, however, so look for involvement outside the respiratory system.

Asthma is a disease confined to the small airways, while anaphylaxis can affect the entire airway, so the presence of a wheeze doesn't differentiate between them. Revisit the patient's airway: ask about swelling of the tongue, lips and face. Is their voice normal? Any of these changes suggest anaphylaxis and, while continuing to treat for asthma is not contraindicated, it will not retard the progression of anaphylaxis. Mistaking anaphylaxis for asthma is probably the most serious error that could occur in this setting.

Similarly, the erythematous appearance of anaphylaxis can easily be confused with the flushed skin of the hard-working asthmatic. Urticaria and irregular rashes are characteristic of anaphylaxis, however: check carefully. Abdominal symptoms are distinctly related to anaphylaxis and the hypotension caused by widespread vasodilation in anaphylaxis tends to occur early compared to its late presentation and association with alterations of conscious state in asthma. Be suspicious of anaphylaxis if you find a hypotensive, conscious patient with a wheeze.

On careful examination, this patient displays no signs of anaphylaxis.

### Arrhythmia

While arrhythmia is not an obvious differential diagnosis, it is not uncommon for arrhythmias such as tachyarrhythmias to cause shortness of breath. This occurs due to inadequate filling times within the heart, resulting in poor cardiac output and delivery of oxygen to the cells. Even in young patients who don't appear to be obvious candidates for arrhythmia, paramedics should investigate this possibility, as these rhythms are often seen in younger age groups. In addition, some recreational drugs and stimulants such as coffee can result in

## DIFFERENTIAL DIAGNOSIS

### Asthma

Or

- Anaphylaxis
- Arrhythmia
- Pneumothorax
- Pleural effusion
- Chest infection
- Anxiety



arrhythmias that can lead to shortness of breath. In this situation, arrhythmia can easily be dismissed by conducting an ECG.

### Pneumothorax

Pneumothorax is a spontaneous rupture of the lung tissue that allows air into the intrathoracic space and restricts ventilation of a portion of the lung. It is rare in young healthy people, but it should be considered in those who present with respiratory distress following trauma, with chronic lung disease such as cystic fibrosis or with connective tissue disorders such as Marfan's syndrome.

Airway narrowing and bronchoconstriction across both lung fields do not match the pathophysiology of a pneumothorax that, if present, should have been noted during the chest auscultation as an area of poor ventilation. This has been excluded in this patient.

### Pleural effusion

The accumulation of fluid in the intrathoracic space can occasionally present with acute respiratory distress symptoms. By compressing an area of the lung the effusion reduces the space available for gas exchange. Rapidly-forming effusions can be the first sign of lung or chest tumours so shouldn't be excluded outright in any patient.

The airway narrowing and bronchoconstriction noted in this patient as wheezing across both lung fields does not match the pathophysiology of a pleural effusion, which should have been noted during the chest examination as an area of poor ventilation. Pleural effusion is therefore not likely in this patient.

### Chest infection

In fit, young and otherwise healthy individuals, chest infections should not generate acute respiratory distress in the absence of signs such as a productive cough, general malaise and fever. Coughing is typical in acute asthma but it is usually non-productive and tends to commence quickly along with the onset of the other symptoms. In a chest infection the presence of abnormal lung sounds is also usually isolated to the area of infection and is therefore rarely perfectly bilateral. Chest infection is therefore excluded in this patient.

#### PRACTICE TIP

Are there signs of bronchoconstriction and small airway occlusion? Yes!  
Are there signs of anaphylaxis other than the above? No  
Are the symptoms affecting the patient's ability to ventilate normally? Yes  
Treat as asthma.

### Anxiety

Anxiety can lead to hyperventilation, which a patient with an asthma history can interpret as an asthma attack. Simultaneously, asthma and chest tightness lead to anxiety and promote hyperventilation. This is probably the most difficult and dangerous diagnosis to exclude. While anxiety and asthma share a number of signs and symptoms, the presence of airway narrowing is limited to asthma. The presence of a wheeze, the prolonged expiratory phase and the use of accessory muscles are all linked to hyperinflated bronchioles that need to be treated. This patient may indeed be anxious about her exams, but that doesn't preclude her from having asthma.

3

## TREAT

### Emergency management

Treatment is aimed at improving the efficiency of ventilation and relieving the bronchospasm and inflammatory response symptoms of asthma (see Box 17.3). What changes as the severity level increases is the speed and level of intervention required to achieve these goals.

## BOX 17.3 Principles of management

**Asthma**

- Improve the efficiency of ventilation for maximum gas exchange
- Provide maximum inspired oxygen for patients with impaired ventilation
- Reduce bronchospasm
- Reduce mucosal oedema and mucus production

**Position**

It is often difficult to assess the severity of a patient's respiratory distress immediately, but patients in extreme difficulty will place themselves in the best position to mechanically ventilate. This means you may find severe asthmatic and COPD patients standing with their arms anchored on tables or walls. You may have to adapt your assessment to the patient's position rather than trying to move them to your preferred position. As a general rule, and provided it is safe to do so, assess and initiate treatment of respiratory patients in the position you find them.

**Bronchodilators****Short-acting beta-adrenergic agonists**

Binding of adrenaline to bronchial smooth muscle causes decreased levels of intracellular calcium and subsequent muscle relaxation. Short-acting beta-adrenergic agonists (SABAs) such as salbutamol have become the cornerstone of acute asthma management (see Table 17.1). The mode of delivery and dosing varies between sufferers, their carers and ambulance services but 10 mg of salbutamol delivered via a nebuliser mask for as long as the patient remains symptomatic is relatively standard (see Box 17.4). For patients with mild symptoms, the use of metered-dose inhalers can reduce the onset of side effects (tremor, tachycardia, arrhythmias, hyperglycaemia) while providing adequate relief, but moderate to severe asthmatics will generally require higher doses. Most of the modern SABAs have been manufactured to bind specifically with the beta-2 ( $\beta_2$ ) sites present on bronchial smooth muscle, but some agencies still administer adrenaline via a nebuliser. This is currently being debated due to the potential for adverse side effects from the alpha and beta-1 ( $\beta_1$ ) properties of this drug.

The effectiveness of inhaled SABAs depends on the patient ventilating sufficiently to deliver the drug to the required site. Provided this occurs there is no benefit to administering SABAs via the IM or IV route, but in cases where the patient has insufficient tidal volume for adequate delivery, IM adrenaline (300–600 mcg) and/or IV salbutamol (250 mcg with 125 mcg repeats five-

TABLE 17.1 Treatments to consider for mild, moderate, severe and life-threatening asthma

Treatment	Mild	Moderate	Severe	Life-threatening/ near-fatal
Oxygen	Yes	Yes	Yes	Yes
Nebulised SABA	Yes	Yes	Yes	Yes
Nebulised ipratropium bromide	No	No	Yes	No
IV salbutamol	No	No	Yes	Yes
Steroids	No	Yes	Yes	Yes
Magnesium sulphate ( $\text{MgSO}_4$ )	No	No	Yes	Yes
IM adrenaline	No	No	Yes	Yes
IPPV/intubation	No	No	No	Yes

Source: Adapted from QAS (2011) and Holly & Boots (2009).

## BOX 17.4 Medications

A well-documented management plan adhered to by the patient and regularly updated with a GP forms the mainstay of treatment for asthma. Plans centre on avoiding triggers, appropriate use of preventer and relieving medications and a pre-established point where steroids are introduced. Unfortunately, with some asthma medications now available without prescription and the breakdown of the traditional GP–patient relationship, it is not uncommon for patients to have a large range of puffers and turbuhalers including:

- two salbutamol puffers under different trade names (Ventolin, Airomir, Asmol)—colour: blue; class: reliever
- ipratropium (Atrovent)—colour: white with a green cap; class: reliever (as it works by a different mechanism of action it is not coloured blue)
- fluticasone (Flixotide)—colour: orange; class: corticosteroid preventer
- ciclesonide (Alvesco)—colour: orange; class: corticosteroid preventer
- beclomethasone (QVAR)—colour: brown; class: corticosteroid preventer
- budesonide (Pulmicort)—colour: brown; class: corticosteroid preventer
- salmeterol (Serevent)—colour: green; class: symptom controller
- eformoterol (Oxis)—colour: green; class: symptom controller
- seretide (Fluticasone and Salmeterol)—colour: purple; class: long-acting reliever
- symbicort (Budesonide and Eformoterol)—colour: red; class: long-acting reliever.

Taking medication in the same colour class can effectively be double-dosing.

minutely to a maximum of 1 mg) should be administered. There is no strong evidence supporting the use of IM adrenaline as historically most clinicians have moved directly to IV salbutamol, but its effectiveness and safety in the management of anaphylaxis suggest it should be used if IV access is not available (or delayed) and the inhaled route is not sufficient.

IM adrenaline administration in patients who are deteriorating despite inhaled SABA therapy is now widely used by Australian ambulance services. The drug reaches therapeutic thresholds quickly (when given into large muscle masses) and has very few side effects when administered by this route.

IV adrenaline to support blood pressure in asthmatic patients compromised by high intrathoracic pressure is an established treatment. The synergistic bronchodilation effects of adrenaline have seen a number of ambulance services replace IV salbutamol infusions with adrenaline infusions.

#### Anticholinergics

Bronchial smooth muscle is innervated by the parasympathetic nervous system. Administration of anticholinergic agents that block the transmission of nerve impulses to the bronchial muscle is effective in reducing bronchospasm. Adding 500 mcg of ipratropium bromide to nebulised SABA is recommended for moderate-to-severe asthma patients. The combination of a SABA and an anticholinergic has proved to be more effective than nebulised  $\beta_2$  agonists alone in severe asthma (National Asthma Council of Australia, 2006). With a four-hour therapeutic range, anticholinergic agents such as ipratropium bromide are generally a once-only administration by paramedics.

#### Aminophylline

By reducing cellular phosphodiesterase activity, aminophylline is an effective smooth muscle relaxant and subsequent bronchodilator that has been shown to be beneficial in severe asthma (Stanley & Tunnicliffe, 2008). However, concerns over side effects (including bradycardia VF and seizures) and a narrow therapeutic range that requires regular monitoring have seen its use decrease in the paramedic setting over the past two decades.

#### Magnesium

In contrast to aminophylline, the administration of a single dose (1–2 mg) of IV or IO magnesium sulphate ( $\text{MgSO}_4$ ) over 20–30 minutes is becoming

a more widely accepted management strategy for asthma and is increasingly used by paramedics. IV  $\text{MgSO}_4$  is supported in the literature as an adjunct to standard treatment for adult patients with severe or life-threatening exacerbation of their asthma (Soong & Chang, 2012). The role of  $\text{MgSO}_4$  in less severe asthma and the role of nebulised  $\text{MgSO}_4$  are less evident due to insufficient evidence (Soong & Chang, 2012). Acting as a smooth muscle relaxant there remain variations in dosages and frequencies across healthcare settings, and the severity of side effects such as hypotension and muscle weakness is not well documented (Stanley & Tunnicliffe, 2008). A second mechanism of action related to its anti-inflammatory properties and involvement in acetylcholine and histamine release may also occur.

### Oxygen

Increasing the fraction of inspired oxygen ( $\text{FiO}_2$ ) of each breath can combat the hypoxia caused by the inadequate tidal volume that acute asthma can cause. Titrating any drug to effect is important and there may be adverse effects associated with administering too much oxygen but this is rare in asthmatic patients. The dynamic nature of asthma and alterations in ventilation efficiency can make it difficult to calculate the  $\text{FiO}_2$ . High-flow oxygen via a Hudson or non-rebreather mask should routinely be administered to all asthmatics with moderate-to-severe symptoms or any compromise of tidal volume. Paramedics may be limited to a single delivery method and concentration.

In the setting of reduced tidal volumes and gas trapping it is vital to acknowledge that while a high  $\text{FiO}_2$  may assist in managing hypoxia, it will not assist in reducing hypercapnia, and these patients may become catatonic due to hypercapnia despite maintaining a high  $\text{SpO}_2$ .  $\text{SpO}_2$  readings may not be predictive of respiratory failure in asthmatics who may return good readings due to gas trapping.

### Steroids

The administration of oral, IM and IV steroids to suppress the inflammatory response is recommended in severe asthma. Although the one- to two-hour onset times for the commonly administered steroids (hydrocortisone, dexamethasone) may exceed the timeframe in which paramedics will be managing asthmatic patients, it appears outcomes are improved when steroids are administered early (Stanley & Tunnicliffe, 2008). Oral administration of corticosteroids appears to work just as effectively as the IM or IV route but may be restricted due to conscious state, breathlessness or simply nausea and/or vomiting (Myers & Tomasio, 2011).

4

## EVALUATE

Evaluating the effect of any clinical management intervention can provide clues to the accuracy of the initial diagnosis. Some conditions respond rapidly to treatment so patients should be expected to improve if the diagnosis and treatment were appropriate. A failure to improve in this situation should trigger the clinician to reconsider the diagnosis.

Although treatment with SABAs to promote bronchodilation treats only one component of the triad that causes airflow restriction (bronchospasm, airway swelling, airway obstruction), they are generally effective in reducing symptom severity and allowing the patient to maintain an adequate tidal volume. Combined with the other inhaled bronchodilators you would expect to see improvement in a patient within 5 minutes. With medication delivered via the nebuliser route this is largely dependent on the patient inhaling sufficiently to deliver the drug to the affected sites in the lung. Misting of the nebuliser mask to the outside environment while the patient is inhaling suggests an inspiratory flow insufficient for drug delivery. In this case the

patient was maintaining a tidal volume that allowed delivery of the medication and the degree of bronchospasm reduced.

Signs of improvement include:

- reduced work of breathing (5–10 minutes)
- increased tidal volume (5–10 minutes)
- respiratory rate may increase as the patient is now able to ventilate more easily (look for the I:E ratio returning towards normal) and to correct hypercapnia
- volume and length of wheezes may actually increase as the amount of air-flow is increased
- coughing may become more apparent as the patient is able to generate higher expiratory flows
- heart rate may remain tachycardic due to the  $\beta_1$  effects of the SABAs.

Critically, the administration of bronchodilators has no effect on the underlying inflammatory process or the chronic airway changes, and the symptoms may return once the bronchodilators are metabolised. Regardless of the degree of improvement this patient must be transported to hospital.

## Ongoing management

### Non-invasive ventilatory support

Continuous positive airway pressure (CPAP) is widely used to provide ventilatory support in diseases such as acute pulmonary oedema and COPD but its role in asthma remains uncertain despite promising studies stretching back four decades (Soroksky et al., 2010; Shivaram et al., 1987). Although it may appear paradoxical to apply additional pressure to a patient already struggling to exhale, the improved outcomes appear to be related to reducing the work of inspiration despite increasing airway resistance airflow during expiration (Soroksky et al., 2010). A number of small studies now suggest that this management strategy may be effective, but identifying which patients will benefit and whether biphasic positive airway pressure (BiPAP) is a better option (Brandao, Lima & Filho, 2009) are still unclear. The lack of a portable system that offers the degree of adjustment required to manage asthmatic patients is likely to restrict this mode of treatment to hospitals for the foreseeable future.

### Heliox

The use of helium and oxygen gas (heliox) for asthma management lacks substantial evidence to support its use and most of the promising studies are limited by small numbers (Reuben & Harris, 2004). As such it is used inconsistently across EDs. The gas has a similar viscosity to air but a much lower density and may maintain

a laminar flow and therefore reduce airway resistance.

### Invasive ventilatory support

Patients who do not respond to pharmacological management and are unable to maintain adequate tidal volume will eventually lose consciousness. Attempting to ventilate this group of patients using bag-valve masks is difficult as the high pressures required to overcome the airway resistance may force the air between the mask and the face, or into the stomach. As a consequence intubation of the trachea is indicated in these patients as the best means of maintaining ventilation.

### Antibiotics

The prevalence of chest infections in acute asthma exacerbations is substantial but they are primarily viral and therefore not appropriate for antibiotic treatment (Teichtahl et al., 1997). They are usually restricted to patients displaying symptomology consistent with pneumonia or a bacterial infection.

### In-hospital tests

While sequential tests of values such as FEV<sub>1</sub> can be useful to determine the effect of treatment, most respiratory tests require the patient to be only in mild distress in order to participate. As a result these tests are usually withheld until after the acute phase of the asthma episode. Similarly, routine blood tests for inflammatory markers are not usually conducted in the ED. Chest x-rays and ECGs are usually only indicated if there is a suspicion of other conditions underlying the respiratory distress.



## Follow-up

Following an acute episode of asthma patients may undergo a series of respiratory tests, allergy-sensitivity tests and a medication review before discharge. This should be followed up with an appointment with the patient's GP to determine the effectiveness of the medications in managing both acute and chronic changes to the airway. The aims of follow-up include to:

- establish or review an asthma management plan with the patient
- investigate factors leading to the acute episode (e.g. medication compliance issues [very common])
- review trigger factor/s with possible allergy testing
- review medications—for example, is the patient taking only relievers rather than preventers, symptom controllers and steroids; and has the use of these medications increased significantly over the last month/s?

## Long-term impact

For years the primary role of the paramedic with regard to asthma patients has been emergency management and transport to definitive care for the condition. Today paramedics responding to asthmatic patients also have the opportunity to recognise patients who are not effectively managing their disease and engage them in the wider health system. In addition they have the opportunity to educate asthma sufferers and their carers as to the appropriate responses to acute asthma. By doing this we can minimise the effects this disease has on the individual and the wider community, with the goal being prevention of morbidity and mortality in what has become an epidemic of industrialised society.

## CS CASE STUDY 2

### Case 11442, 2012 hrs.

**Dispatch details:** A 30-year-old male in severe respiratory distress with a history of asthma.

**Initial presentation:** On a cold winter's night the ambulance crew locate the patient in the lounge of his fourth-floor apartment. He is sitting upright on the edge of his chair. He has both palms on the table in front of him with his arms extended. He is conscious and talking.

## 1 ASSESS

**2024 hrs Chief complaint:** 'My asthma ... has been ... playing up ... for a couple of weeks. I've got ... a national tryout ... for hockey ... coming up ... and throughout training ... I couldn't catch ... my breath. My puffers ... aren't working'.

**2024 hrs Vital signs survey:** Perfusion status: HR 128, BP 158/91 mmHg, skin cool and pale.

Respiratory status: anxious, struggling to breathe, respiratory rate 30, SpO<sub>2</sub> 92%, air entry, L = R, inspiratory and expiratory wheeze all lung fields, use of accessory muscles (tripod position), speaking in short sentences, prolonged expiratory phase, complains of shortness of breath.

Conscious state: GCS = 15, anxious.

**2027 hrs Pertinent hx:** The patient's asthma (diagnosed at 6 years of age, with improvement during his teen years) is normally well-controlled with one

mild exacerbation per year usually associated with a chest infection. For the last 2 months he has been training hard to get in shape for a national hockey team but since the start of winter he has been suffering progressively worsening episodes of asthma that have affected his ability to train. He has seen a number of GPs who have all prescribed steroids and a wide variety of puffers. Due to steroids being disallowed in sport he has been non-compliant with this component of treatment and has been using his reliever medication (primarily salbutamol) more than prescribed.

## 2 CONFIRM

What else could it be?

### Anxiety

Although hyperventilation can cause tetany of skeletal muscles, this does not affect bronchial smooth muscle. The question here is not, 'Is the patient anxious?', as shortness of breath will cause anxiety, but 'Does the patient have bronchospasm?' If yes, it needs to be treated. Asking the patient to relax will not relieve the inflammatory cause affecting his lungs.

### Anaphylaxis

The hyperreactive immune nature of both asthma and anaphylaxis require paramedics to clearly distinguish between the two before commencing treatment. The absence of upper airway symptoms, erythematous rash, urticaria and any gastrointestinal symptoms suggests the condition is definitely confined to the lungs. His relatively high blood pressure points away from any cardiovascular involvement.

### Cardiac arrhythmia

While cardiac arrhythmias such as supraventricular tachycardia can be associated with shortness of breath, this is almost always because of poor cardiac output and perfusion. The blood pressure alone would suggest this is not a problem but an ECG recording will confirm it. Again, the primary problem is the increased work of breathing and there is no link between arrhythmia and bronchospasm.

### Chest infection

An exacerbation of this patient's asthma caused by an acute infection is possible and likely. Inflammation associated with the infection could present as a wheeze due to either primary oedema or triggering bronchospasm. The absence of a productive cough or a temperature means relatively little in the acute phase. The patient's asthma has been worsening for 2 months, making the lack of any episode of a temperature, productive cough or other symptoms of infection an unlikely source of this episode of asthma (and the increase in frequency of associated symptoms). The bilateral wheeze, without any zone of alteration in breath sounds, also indicates the lack of an area of consolidated infection.

## 3 TREAT

**2028 hrs:** The patient is given 10 mg of salbutamol with 500 mcg of ipratropium bromide via a nebuliser mask and supplemental oxygen at 8 L/minute. The crew observe that the mask continually mists into the room air even when the patient tries to inhale. Anticipating that the patient may not improve, they immediately draw up IM adrenaline and prepare to insert an IV.

**2031 hrs:** The patient is still sitting upright but is now staring straight ahead and is no longer responding to verbal requests. He is not speaking.

Perfusion status: HR 154 BPM, sinus tachycardia, BP 150/90 mmHg, skin warm and pink.

### USING THE MNEMONIC DENT

Define:

- What is the patient's main presenting problem?
- Is this asthma?
- Does it fit the definition?

Explore your hypothesis and try to narrow it down. What else could it be?

- Anxiety
- Anaphylaxis
- Cardiac arrhythmia
- Chest infection

Respiratory status: catatonic, struggling to breathe, RR 30 BPM, SpO<sub>2</sub> 92%, air entry, L = R, poor tidal volume, inspiratory and expiratory wheeze all lung fields, use of accessory muscles (tripod position), not speaking, prolonged expiratory phase.

Conscious state: GCS = 13.

**2031 hrs:** Aware that the patient is deteriorating and not ventilating sufficiently to deliver the inhaled beta-agonist to the smooth muscle of the lungs, the crew administer 0.3 mg of adrenaline IM to his right thigh while establishing IV access. As with anaphylaxis, IM adrenaline has little impact on heart rate or blood pressure and although adrenaline is generally considered to treat perfusion, in this case it is being given as a  $\beta_2$ -agonist to promote bronchodilation. The alpha and  $\beta_1$  effects are not important in this setting.

**2033 hrs:** The patient has failed to respond to the IM adrenaline and has slumped forwards onto the table. The crew lower him to the floor and obtain IV access.

Perfusion status: strong pulse, HR 154 BPM, sinus tachycardia, BP 130/90 mmHg, skin warm and pink.

Respiratory status: unconscious, increased work of breathing, RR 30 BPM, SpO<sub>2</sub> 92%, air entry, L = R, poor/no tidal volume, quiet wheezes all lung fields, use of accessory muscles, not speaking, prolonged expiratory phase.

Conscious state: GCS = 3.

**2035 hrs:** The crew have administered magnesium by the IV route and prepare a SABA or inotrope infusion to commence once the bolus doses have been exhausted. The patient remains unconscious and is difficult to ventilate due to high airway pressures. The crew attempt to ventilate at half the normal respiratory rate (5 BPM) and allow a prolonged expiratory phase in an attempt to reduce the hyperinflation. (Some ambulance services recommend assisting the expiratory phase by applying gentle pressure to the rib cage in the mid-axillary line. There is no high-level evidence to support the practice and there is a physiological reason to be cautious because the occlusion of the airway will exacerbate gas trapping [West, 2012], but anecdotal reports have seen it remain in some state guidelines.) The patient's blood pressure and pulse remain adequate.

**2038 hrs:** There is no improvement in the patient; he is failing to respond to the IV medication and the crew are unable to effectively ventilate him using a BVM due to the high airway pressures required.

Perfusion status: HR 154 BPM, sinus tachycardia, BP 130/90 mmHg, skin warm and pink.

Respiratory status: unconscious, struggling to breathe, RR 30 BPM, SpO<sub>2</sub> 92%, air entry, L = R, poor/no tidal volume, quiet wheezes all lung fields, use of accessory muscles, not speaking, prolonged expiratory phase.

Conscious state: GCS = 3.

**2040 hrs:** The best method of ventilating this patient is via an endotracheal tube as it reduces the dead space, provides an FiO<sub>2</sub> of 100% and allows for higher pressure to be used without the risk of inflating the stomach. Vitally, the crew must allow the patient adequate time to expire before trying to force any volume of air into his lungs. The use of an endotracheal tube in this setting risks both barotrauma to the patient (from the high inspiratory pressures) and complications associated with inserting the tube. Asthmatic patients who have become unconscious and require intubation are likely to have a degree of jaw tone and the use of short-acting paralysing agents combined with sedation is likely to provide a better view of the vocal cords.

### PRACTICE TIP

*Check for a pulse!* Has the patient lost consciousness because hyperinflation has occluded blood returning to the heart? If this is the case, a period of apnoea may allow gas to escape and attempting to force inspiration will only worsen the condition. If the patient remains pulseless after 1 minute of apnoea, there is little alternative to commencing the cardiac arrest guidelines, but a spontaneous tension pneumothorax should also be considered.

### PRACTICE TIP

Some ambulance services do not promote the use of paralysing agents in a patient with established respiratory distress. The view is that the patient already has a compromised ventilator effort and paralysing them only removes what little effort they already have.

While maintaining doses of SABA and other bronchodilators, the crew administer 125 mg of suxamethonium (short-acting paralysing agent) with 50 mcg of fentanyl and 3.5 mg of midazolam to achieve a successful intubation.

**2043 hrs:** The patient remains unconscious but has not deteriorated further. The crew can move approximately 250 mL of air in and out of his lungs with each ventilation but the prolonged expiratory phase allows for only 6–8 BPM. The patient's blood pressure remains good so there is no evidence yet of the high airway pressures causing a pneumothorax.

Perfusion status: There is a pulse, HR 154 BPM, sinus tachycardia, BP 130/90 mmHg, skin warm and pink.

Respiratory status: Unconscious, difficult to ventilate, RR 30 BPM, SpO<sub>2</sub> 92%, EtCO<sub>2</sub> = 55 mmHg, air entry, L = R, poor/no tidal volume, quiet wheezes all lung fields, use of accessory muscles, not gagging on the endotracheal tube, prolonged expiratory phase allowed.

Conscious state: GCS = 3.

With the patient being ventilated as effectively as possible, infusions supporting the management of bronchospasm and IV steroid having been administered, the priorities are conceptually simple (although clinically challenging):

- **Airway.** Maintain the endotracheal tube with the use of sedation. As unconscious asthmatic patients are ventilated effectively their conscious state may improve and they may require increased sedation to maintain the tube placement. Increasing sedation doses may impact on blood pressure, which may then need inotropic support.
- **Breathing.** Reducing bronchospasm to reduce airway resistance remains the focus. The crew should maintain ongoing doses of bronchodilators according to local guidelines and continue to allow for a prolonged expiratory phase. If the patient's own respiratory efforts are compromising effective ventilation via the endotracheal tube the crew should consider a long-acting paralysing agent according to local guidelines. This will require an increased vigilance of sedation doses as pharmacologically paralysed patients cannot indicate inadequate sedation.
- **Circulation.** Sudden falls in blood pressure can occur due to either recurrent gas trapping or the formation of a tension pneumothorax. The crew should remain vigilant for both. Inadequate blood pressure should be supported with inotropic infusions.

4

## EVALUATE

Evaluating the effect of any clinical management intervention can provide clues to the accuracy of the initial diagnosis. Some conditions respond rapidly to treatment so patients should be expected to improve if the diagnosis and treatment were appropriate. A failure to improve in this situation should trigger the clinician to reconsider the diagnosis.

This patient's deterioration is not an indication of a misdiagnosis but rather that his lack of tidal volume has resulted in the nebuliser drugs not being inhaled (as indicated by the constantly misting mask). This result is not unexpected and the crew need to use another route to treat the underlying cause.

## Future research

Leukotriene receptor antagonists have been proven to reduce the amount of  $\beta_2$  agonists required in severe asthma. They have been found

to be less effective than steroids but have fewer side effects and can be used as a steroid limiter, particularly in children (National Asthma Council Australia, 2010). Their use in adults requires

ongoing research. The role of parasitic worms in controlling inflammatory diseases such as asthma is also being investigated (Zaccone, 2006).

## Summary

As paramedics we often deal with the whole spectrum of asthma, from parents seeking advice

for children who have a cough to cardiac arrests associated with the condition. Many paramedics will rarely see severe or near-fatal asthma, but with 416 deaths occurring from this condition in Australia in 2010 (National Asthma Council Australia, 2013) it is prudent to be able to recognise the condition in all of its iterations.

## References

- Amatoury, J., Kairaitis, K., Wheatley, J. R., Bilston, L. E., & Amis, T. C. (2010). Onset of airflow limitation in a collapsible tube model: impact of surrounding pressure, longitudinal strain, and wall folding geometry. *Journal of Applied Physiology*, 109.
- Brandao, D. C., Lima, V. M., & Filho, V. G. (2009). Reversal of bronchial obstruction with bi-level positive airway pressure and nebulization in patients with acute asthma. *Journal of Asthma*, 46.
- Cox, G. (1995). Glucocorticoid treatment inhibits apoptosis in human neutrophils. *Journal of Immunology*, 154, 4719.
- Craft, J., Gordon, C., & Tiziani, A. (2010). *Understanding Pathophysiology*. Sydney: Elsevier.
- Curtis, K., & Ramsden, C. (2011). *Emergency and Trauma Care for Nurses and Paramedics*. Sydney: Elsevier.
- Des Jardins T., Burton G. G. Clinical manifestations and assessment of respiratory disease. 5th edn. St Louis: Mosby; 2006.
- Donald, M., & Paterson, B. (2006). End tidal carbon dioxide monitoring in prehospital and retrieval medicine: a review. *Emergency Medicine Journal*, 23(9), 728–730.
- Gavriely, N., & Grotberg, J. B. (1988). Flow limitation and wheezes in a constant flow and volume lung preparation. *Journal of Applied Physiology*, 64, 17–20.
- Gavriely, N., Shee, T. R., Cugell, D. W., & Grotberg, J. B. (1989). Flutter in flow-limited collapsible tubes: a mechanism for generation of wheezes. *Journal of Applied Physiology*, 66(5), 2251–2261.
- Holly, A. D., Boots, R. J. (2009). Review article: management of acute severe and near-fatal asthma. *Emergency Medicine Australasia*, 21(4).
- McCance, K., & Huether, S. (2014). *Pathophysiology: The Biologic Basis for Disease in Adults and Children* (7th ed.). Philadelphia: Mosby.
- Murphy, D. M., & O'Byrne, P. M. (2010). Recent advances in the pathophysiology of asthma. *CHEST*, 137(6), 1417–1426.
- Myers, T. R., & Tomasio, L. (2011). Asthma: 2015 and beyond. *Respiratory Care*, 56(9).
- National Asthma Council Australia. (2006). *Asthma Management Handbook*. Retrieved 23 May 2013 from [www.nationalasthma.org.au/handbook](http://www.nationalasthma.org.au/handbook).
- National Asthma Council Australia. (2010). Leukotriene receptor antagonists in the management of childhood asthma. Retrieved 24 May 2013 from [www.nationalasthma.org.au/uploads/content/241-2010\\_ltra\\_info\\_paper.pdf](http://www.nationalasthma.org.au/uploads/content/241-2010_ltra_info_paper.pdf).
- National Asthma Council Australia. (2013). Asthma mortality statistics. Retrieved 22 May 2013 from [www.nationalasthma.org.au](http://www.nationalasthma.org.au).
- Pasterkamp, H., Kraman, S. S., & Wodicka, G. R. (1997). Respiratory sounds: advances beyond the stethoscope. *American Journal of Respiratory Critical Care Medicine*, 156(3).
- Queensland Ambulance Service (QAS). (2011). *Ambulance Service Clinical Practice Manual*. Queensland Government.
- Reuben, A. D., & Harris, A. R. (2004). Heliox for asthma in the emergency department: a review of the literature. *Emergency Medicine Journal*, 21.
- Rodriguez-Roisin, R. (1997). Acute severe asthma: pathophysiology and pathobiology of gas exchange abnormalities. *European Respiratory Journal*, 10, 1359–1371.
- Saetta, S., & Turato, G. (2001). Airway pathology in asthma. *European Respiratory Journal*, 18(4), 18s–23s.
- Shivaram, U., Donath, J., Khan, F. A., & Juliano, J. (1987). Effects of continuous positive airway pressure in acute asthma. *Respiration*, 52(3).
- Soong, W., & Chang, Y. (2012). Magnesium sulfate for acute asthma in adults: a systematic literature review. *Asia Pacific Allergy*, 2(1), 76–85.
- Soroksky, A., Klinowski, E., Ilgyev, E., Mizrahi, A., Miller, M., Ben Yehuda, T. M., et al. (2010). Noninvasive positive pressure ventilation in acute asthmatic attack. *European Respiratory Review*, 19(115).
- Spence, D. P., Graham, D. R., Jamieson, G., Cheetham, B. M., Calverley, P. M., & Earis, J. E. (1996). The relationship between wheezing and lung mechanics during methacholine-induced bronchoconstriction in asthmatic subjects. *American Journal of Respiratory Critical Care Medicine*, 154(2), 290–294.
- Stanley, D., & Tunnicliffe, W. (2008). Management of life-threatening asthma in adults. *Continuing Education in Anaesthesia, Critical Care & Pain*, 8(3), 95–99.
- Stather, D. R., & Stewart, T. E. (2005). Clinical review: mechanical ventilation in severe asthma. *Critical Care*, 9(6).
- Sutherland, E. R., & Martin, R. J. (2007). Asthma and atypical bacterial infection. *CHEST*, 132(6).
- Teichtahl, H., Buckmaster, N., & Pertnikovs, E. (1997). The incidence of respiratory tract infection in adults requiring hospitalization for asthma. *CHEST*, 112(3).
- West, J. (2012). *Respiratory Physiology: The Essentials* (9th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Wyka, K. A., Mathews, P. J., & Clark, W. F. (2011). *Foundations of Respiratory Care* (2nd ed.). USA: Delmar Cengage Learning.
- Zaccone, P., Fehervari, Z. et al. (2006). Parasitic worms and inflammatory diseases. *Parasite Immunology*, 28(10).