FISIOLOGI LINGKUNGAN DI DAERAH TINGGI

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As humans have ascended to higher and higher altitudes in aviation, mountain climbing, and space vehicles, it has become progressively more important to understand the effects of altitude and low gas pressures on the human body. This chapter deals with these problems, as well as acceleratory forces, weightlessness, and other challenges to body homeostasis that occur at high altitude and in space flight.

High-altitude illness

The term *high-altitude illness* is reserved for three unique cerebral and pulmonary maladies that develop over hours to days at high altitude as a result of acute exposure to hypobaric hypoxia: acute mountain sickness (AMS), high-altitude cerebral edema (HACE), and high-altitude pulmonary edema (HAPE). Despite the fact that high-altitude illness is preventable, AMS/HACE and HAPE remain common consequences of rapid ascent. Worldwide, millions of lowland residents who make the journey to high altitudes for recreational, religious, economic, and military purposes are at risk for experiencing some degree of high-altitude illness. As the global popularity of recreating and residing at high altitude continues to increase, practitioners must become familiar with the physiologic challenges and dangers associated with acute exposure to high altitude.

PO^2

EFEK TEKANAN OKSIGEN YANG RENDAH TERHADAP TUBUH

Tekanan barometer di berbagai ketinggian

Table 43-1. Effects of Acute Exposure to Low Atmospheric Pressures on Alveolar Gas Concentrations and Arterial Oxygen Saturation*

Altitude (ft/meters)	Barometric Pressure (mm Hg)	P_{O_2} in Air (mm Hg)	Breathing Air			Breathing Pure Oxygen		
			P_{CO_2} in Alveoli (mm Hg)	*P_{O_2} in Alveoli (mm Hg)*	*Arterial Oxygen Saturation (%)*	*P_{CO_2} in Alveoli (mm Hg)*	*P_{O_2} in Alveoli (mm Hg)*	*Arterial Oxygen Saturation (%)*
0	760	159	40 (40)	104 (104)	97 (97)	40	673	100
10,000/3048	523	110	36 (23)	67 (77)	90 (92)	40	436	100
20,000/6096	349	73	24 (10)	40 (53)	73 (85)	40	262	100
30,000/9144	226	47	24 (7)	18 (30)	24 (38)	40	139	99
40,000/12,192	141	29				36	58	84
50,000/15,240	87	18				24	16	15

*Numbers in parentheses are acclimatized values.

Ketinggian ↑ tekanan barometer ↓→penyebab dasar hipoksia pada tempat tinggi

Tekanan parsial PO^2 ↓ →tekanan selalu tetap dari waktu

Table 1
Altitude, barometric pressure, ambient PO_2, and PIO_2

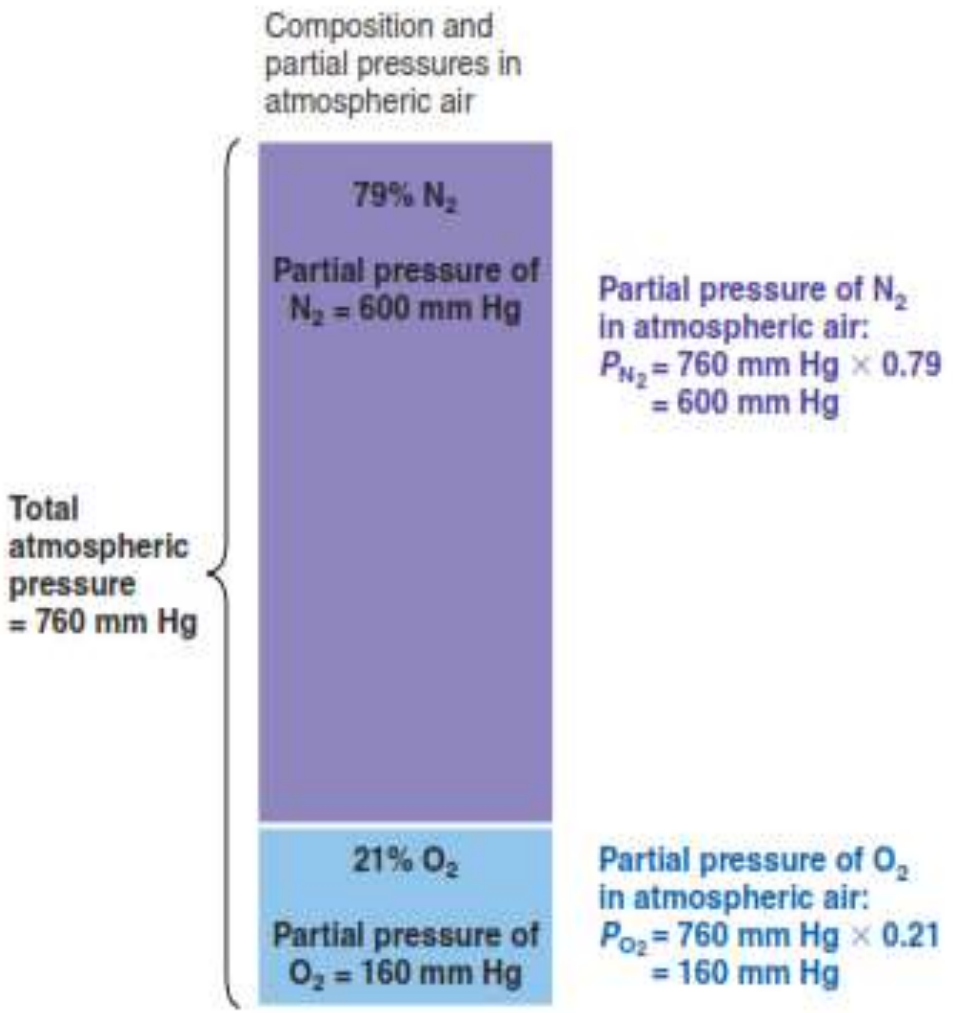
Altitude (m)	Altitude (ft)	Barometric pressure (mm Hg)	Ambient PO_2 (mm Hg)	PIO_2 (mm Hg)
0	0	759.6	159.1	149.1
1000	3281	678.7	141.2	132.2
2000	6562	604.5	124.9	116.7
3000	9843	536.9	110.1	102.5
4000	13,123	475.4	96.9	89.7
5000	16,404	419.7	84.8	78.0
6000	19,685	369.4	79.1	67.5
7000	22,966	324.2	67.8	58.0
8000	26,247	283.7	59.3	49.5
8850	29,035	252.7	52.9	43.1

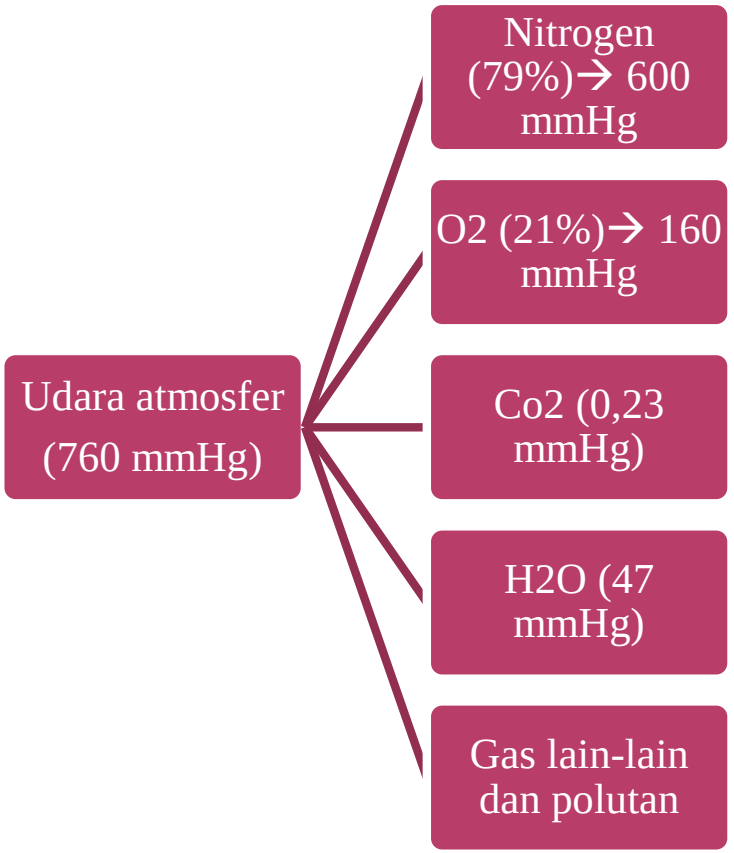
The partial pressure of oxygen (PO_2) in ambient air and inspired air (PIO_2) fall nearly exponentially as a function of increasing altitude and falling barometric pressure.

Adapted from Hackett PH, Roach RC. High-altitude medicine. In: Auerbach PS, editor. Wilderness medicine. Philadelphia: Mosby; 2001. p. 2–43; with permission.

P_{Gas} TEKANAN PARSIAL

- Tekanan yg ditimbulkan secara independen oleh masing-masing gas dlm suatu campuran gas. Semakin besar tekanan parsial suatu gas dlm cairan, semakin banyak gas tersebut terlarut.





PO2 DAN PCO2 ATMOSFER DAN ALVEOLUS

- Komposisi udara alveolus tidak sama dengan komposisi udara atmosfer, karena:

1. Udara atmosfer masuk saluran napas→ pajanan lembap→ jenuh H2O (diencerkan)

PH2O = 47 mmHgA

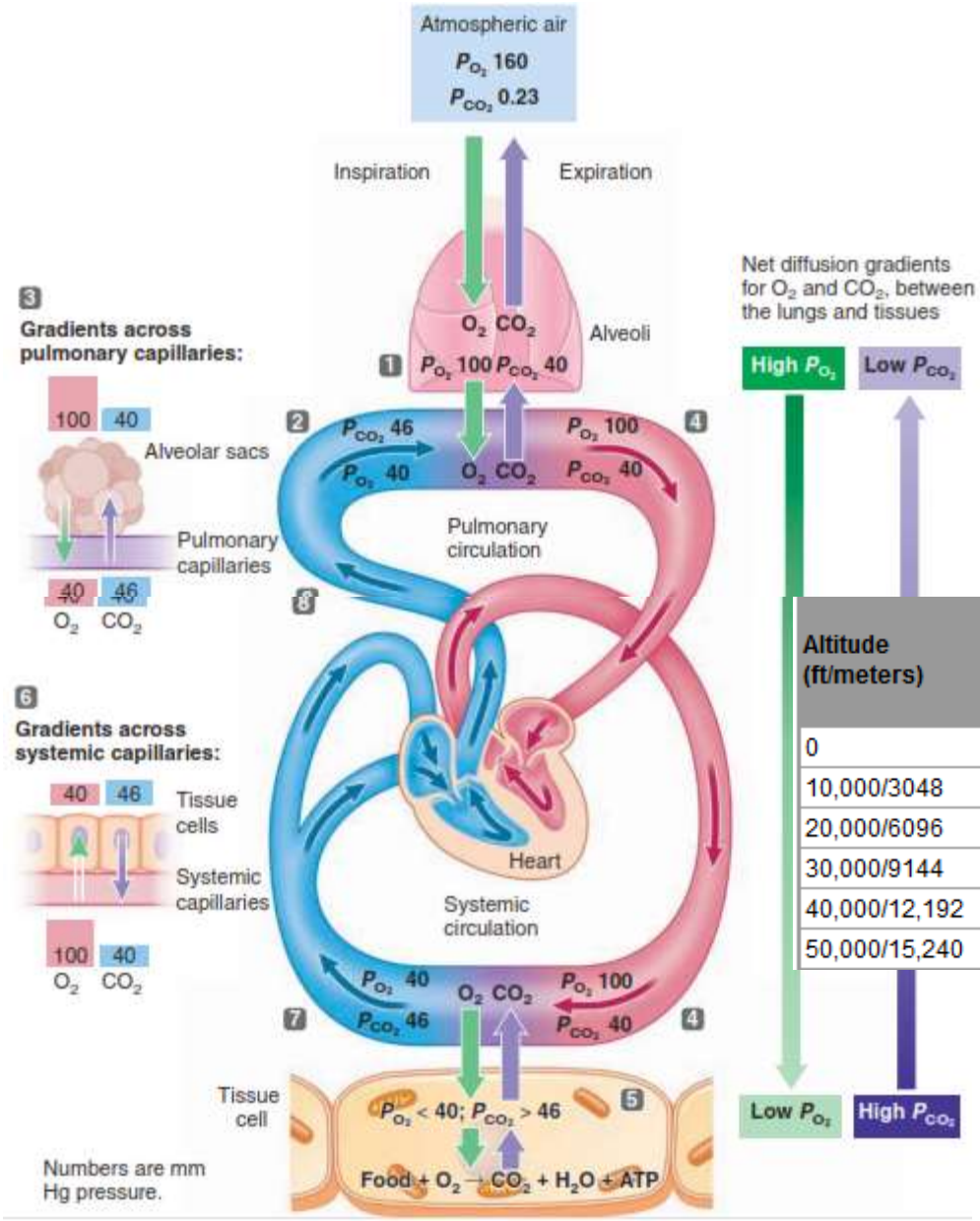
PN2 = 563 mmHg ← 600 mmHg

PO2 = 150 mmHg ← 160 mmHg

PCO2 = 40 mmHg ← 0,23 mmHg

2. Po2 alveolus < Po2 atmosfer→ udara segar yg masuk bercampur dgn sejumlah besar udara lama yg tersisa di paru dan ruang rugi pada akhir ekspirasi sebelumnya.

GRADIEN PO2 DAN PCO2 MENEMBUS KAPILER PARU



Altitude (ft/meters)	Barometric Pressure (mm Hg)	Po_2 in Air (mm Hg)	*Pco_2 in Alveoli (mm Hg)*	*Po_2 in Alveoli (mm Hg)*	*Arterial Oxygen Saturation (%)*
		Breathing Air			
0	760	159	40 (40)	104 (104)	97 (97)
10,000/3048	523	110	36 (23)	67 (77)	90 (92)
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40,000/12,192	141	29			
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Box 1. Categories of high altitude and their physiologic effects

High altitude: 1500 to 3500 m (4921–11,483 ft)

- High-altitude illness common with abrupt ascent to above 2500 m (8202 ft)
- Decreased exercise performance and increased ventilation
- Only minor impairment in arterial oxygen saturation (SaO_2); arterial PO_2 (PaO_2), 55 to 75 mm Hg

Very high altitude: 3500 to 5500 m (11,483–18,045 ft)

- Most common range for severe high-altitude illness
- Abrupt ascent may be dangerous; requires a period of acclimatization
- SaO_2, 75% to 85%; PaO_2, 40 to 60 mm Hg
- Extreme hypoxia may occur during sleep, exercise, and high-altitude illness

Extreme altitude: 5500 to 8850 m (18,045–29,035 ft)

- Progressive deterioration of physiologic function eventually outstrips acclimatization
- Above the highest permanent human habitation
- Abrupt ascent almost always precipitates severe high-altitude illness
- Severe hypoxia and hypocapnia; SaO_2, 58% to 75%; PaO_2, 28 to 40 mm Hg

Data from Hackett PH, Roach RC. High-altitude medicine. In: Auerbach PS, editor. Wilderness medicine. Philadelphia: Mosby; 2001. p. 2–43; with permission.

Box 2. Terminology of medical conditions at high altitude

High-altitude illness

- Acute mountain sickness (AMS)
- High-altitude cerebral edema (HACE)
- High-altitude pulmonary edema (HAPE)

Examples of altitude-related medical problems (partial list)

- High-altitude illness (AMS, HACE, HAPE)
- High-altitude headache
- Peripheral edema
- High-altitude retinopathy
- High-altitude pharyngitis and bronchitis
- Periodic breathing
- Chronic mountain sickness
- Subacute mountain sickness
- Decreased birth weight
- Ultraviolet keratitis
- Hypothermia and frostbite

Examples of altitude-exacerbated conditions (partial list)

- Various congenital and valvular heart diseases
- Primary and secondary pulmonary hypertension
- Symptomatic coronary artery disease
- Poorly compensated congestive heart failure and chronic obstructive pulmonary disease
- Sickle cell disease and trait
- Obstructive sleep apnea
- Urinary retention from benign prostatic hypertrophy
- High-risk pregnancy
- Radial keratotomy

PO^2 ALVEOLAR DI BERBAGAI KETINGGIAN

- **Karbon dioksida dari darah paru ke alveoli dan uap air yg menguap ke dlm udara inspirasi dari permukaan alat pernapasan menurunkan oksigen alveolar**→ mengencerkan dan menurunkan kadar oksigen alveoli.

- Tekanan uap air di dalam alveoli tidak bergantung pada ketinggian dan selama suhu tubuh normal tekanan uap air adalah **47** mmHg.

APAKAH AKLIMATISASI?

Physiologic adjustment to the diminished ambient oxygen pressure of high altitude is known as **acclimatization**.

Other environmental stresses of high altitude include decreased temperature, lower humidity, and increased ultraviolet radiation.

These stresses also have medical consequences.

Conditions arising directly from the high-altitude environment may be termed altitude-related illnesses, whereas pre-existing medical problems made worse by high altitude may be referred to as altitude-exacerbated conditions.

PADA PUNCAK G.EVEREST HANYA ORANG DGN AKLIMATISASI TERBAIK SAJA YG DAPAT BERTAHAN HIDUP SAAT MENGHIRUP UDARA

Gunung Everest (29.028 kaki)

Tekanan barometer 253 mmHg

Tekanan uap air 47 mmHg

Gas-gas lain 206 mmHg

- Teraklimatisasi→ ventilasi meningkat 5x lipat sehingga PCO2 alveolar 7 mmHg, 199 mmHg (O2 dan N) 40~35mmHg

Acclimatization

Ascent to a high altitude requires adjustments that minimize hypoxemia and maintain cellular function despite decreased PO_2. This process is termed *acclimatization*; it is complex, incompletely understood, and involves nearly every organ system. There are limits to compensation for diminished PO_2, and these limits vary markedly among individuals, different human populations, and even animal species. Given sufficient time, most individuals are able to acclimatize up to about 5500 m (18,045 ft). Above this elevation, progressive deterioration outstrips the ability of the body to compensate [1–3].

AKLIMATISASI PO2

Table 43-1. Effects of Acute Exposure to Low Atmospheric Pressures on Alveolar Gas Concentrations and Arterial Oxygen Saturation*

Altitude (ft/meters)	Barometric Pressure (mm Hg)	Po_2 in Air (mm Hg)	Breathing Air		
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40,000/12,192	141	29			
50,000/15,240	87	18			

*Numbers in parentheses are acclimatized values.

Ventilasi alveolar meningkat sekitar 5x pada orang yg teraklimatisasi

Kecepatan udara baru yg masuk pada area alveoli, kantong alveolus, duktus alveolaris, dan bronkiolus respiratorius→ **ventilasi alveolar**

EFEK MENGHIRUP OKSIGEN MURNI TERHADAP PO2 ALVEOLAR DI BERBAGAI KETINGGIAN

Seseorang menghirup **oksigen murni** sbg pengganti udara biasa

Alveoli yg sebelumnya terisi Nitrogen akan terisi oleh oksigen

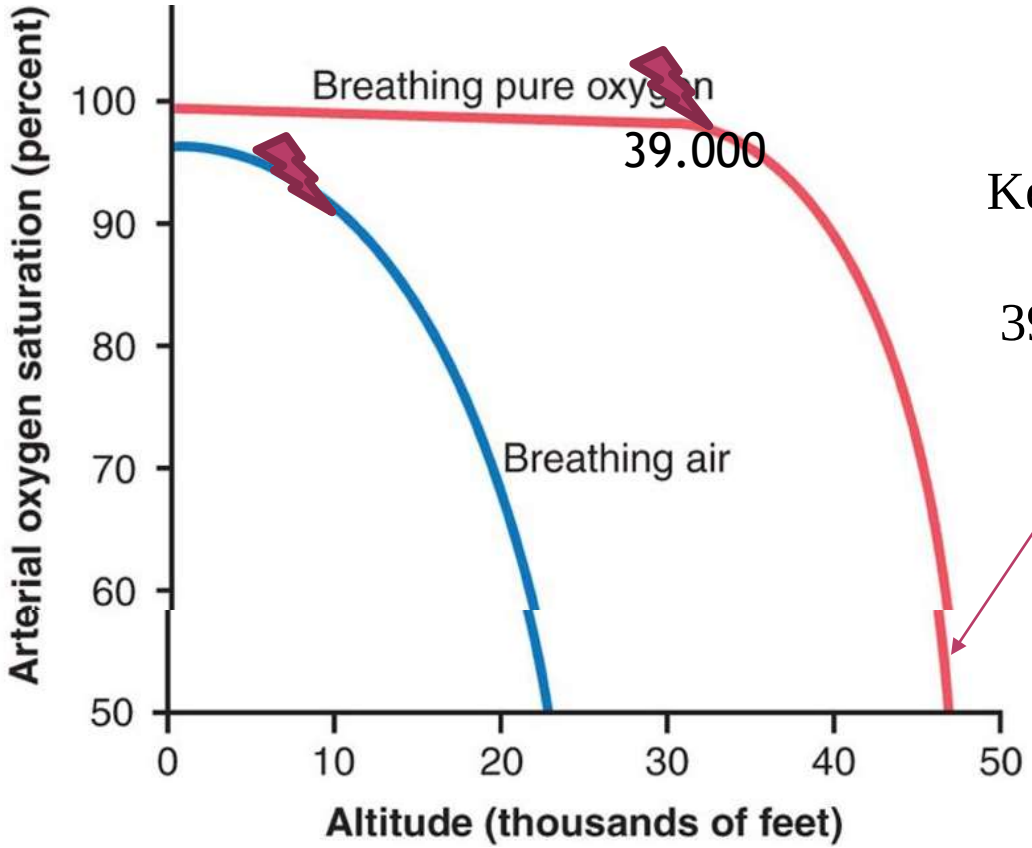
Contoh
kasus pada penerbang:
Ketinggian 30.000 kaki
PO2 alveoli 139 mmHg dengan menghirup oksigen murni→saturasi oksigen 99%
18 mmHg tanpa menghirup oksigen murni→ saturasi oksigen 24%

SATURASI OKSIGEN

- Tingkat oksigen dalam tubuh dapat diukur dengan bantuan berbagai metode. Cara yang paling umum untuk menentukan apakah tingkat kejenuhan oksigen dalam darah adalah dengan memeriksa gas darah arteri dan **oksimeter**.

- Oksimetri adalah sebuah perangkat kecil yang digunakan untuk mengukur kadar oksigen dalam darah.

SATURASI OKSIGEN DI BERBAGAI KETINGGIAN



Pada penerbang:

Ketinggian 30.000 kaki→ PO2 **139** (udara biasa:18) mmHg

39.000 kaki, saturasi O2 tetap diatas 90%

47.000 kaki, saturasi O2 menurun dengan cepat sampai **50% (dapat tetap sadar)**

Pengaruh ketinggian tempat terhadap saturasi oksigen arteri ketika menghirup udara dan menghirup oksigen murni.

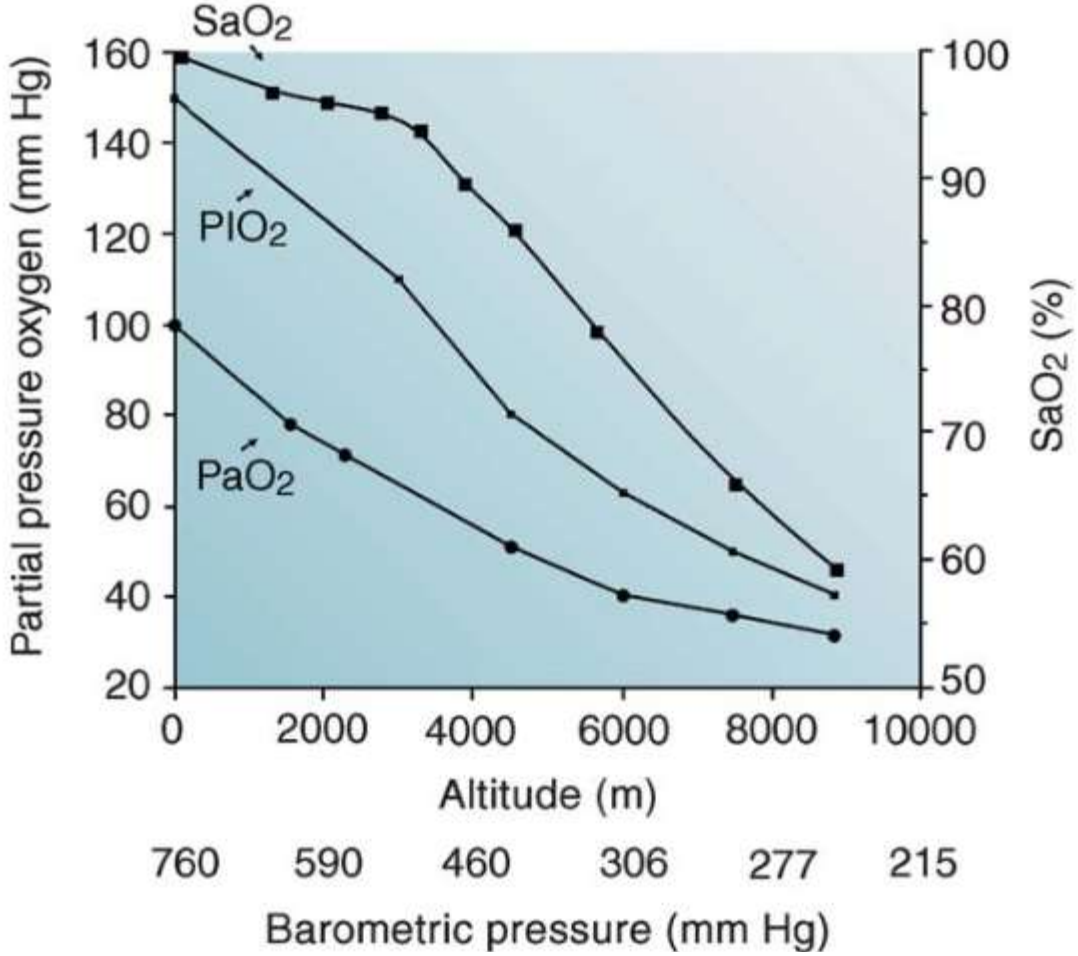


Fig. 1. Relationship of altitude and barometric pressure to PaO_2, PIO_2, and SaO_2. Increasing altitude results in decreased barometric pressure, partial pressure of arterial oxygen (PaO_2), and arterial oxygen saturation (SaO_2). Oxygen saturation is well maintained up to about 3000 m, despite a significant decrease in arterial PO_2. Above this altitude, small changes in arterial PO_2 result in large changes in arterial saturation. (*From* Hackett PH, Roach RC. High-altitude medicine. In: Auerbach PS, editor. Wilderness medicine. Philadelphia: Mosby; 2001. p. 5; with permission.)

"PLAFON" YG DICAPAI KETIKA MENGHIRUP UDARA BIASA OKSIGEN DALAM PESAWAT TANPA ALAT PENGATUR TEKANAN

Menghirup udara biasa hanya mampu terbang 23.000 kaki (nilai batas bagi penerbang yg **sudah** teraklimatisasi)

15.000 kaki batas maksimal bagi penerbang yg **belum** teraklimatisasi

Menghirup oksigen murni mampu terbang hingga ketinggian kurang lebih 47.000 kaki

23.000 kaki (tanpa O2 murni) = 47.000 kaki (dgn O2 murni)→ SaO2 50%

HIPOKSIA

Hypoxemia

Consequent to the reduced PO_2 of ambient air, inspired PO_2 (PIO_2), alveolar PO_2 (PAO_2), arterial blood oxygen (PaO_2), and arterial oxygen saturation (SaO_2) all decrease with progressive ascent and falling barometric pressure (Fig. 1) [1–3]. The cells are impacted by the degree of hypoxemia (low blood oxygen), which can be variable for a given altitude, depending on factors such as compensatory hyperventilation, lung function, and hemoglobin affinity for oxygen. Respiratory depressants, vigorous exertion, hypothermia, and certain pre-existing medical conditions aggravate altitude hypoxemia [4].

Hypoxia Insufficent O_2 at the cellular level

Anemic hypoxia Reduced O_2-carrying capacity of the blood

Circulatory hypoxia Too little oxygenated blood delivered to the tissues; also known as stagnant hypoxia

Histotoxic hypoxia Inability of the cells to use O_2 available to them

Hypoxic hypoxia Low arterial blood P_{O_2} accompanied by inadequate Hb saturation

EFEK AKUT HIPOKSIA

EFEK UTAMA HIPOKSIA

- Menurunnya kecakapan mental
- Menurunkan kemampuan dlm mengambil keputusan, mengingat
- Menurunnya gerakkan motorik terampil

15.000 kaki batas maksimal bagi penerbang yg **belum** teraklimatisasi→ terbang selama 1 jam→ kemampuan mental menurun 50% dari normal→ setelah 18 jam→ turun jadi 20% dari normal.

- Peningkatan ventilasi adalah mekanisme yang efektif untuk menghilangkan hipoksemia

PRINSIP-PRINSIP UTAMA YG TERJADI PADA AKLIMATISASI

Semakin lama (hari-minggu-tahun),makin teraklimatisasi thd PO_2 rendah $\rightarrow$ efek buruk $\downarrow \rightarrow$ bekerja tanpa efek hipoksia

1. Peningkatan ventilasi paru yg cukup besar
2. Peningkatan jumlah sel darah merah
3. Peningkatan kapasitas difusi paru
4. Peningkatan vaskularisasi jaringan perifer
5. Peningkatan kemampuan sel dalam menggunakan oksigen sekalipun nilai PO2 rendah

1. PENINGKATAN VENTILASI PARU YG CUKUP BESAR

RESEPTOR YANG MENGATUR FUNGSI VENTILASI:

Pusat pernapasan

- Medula oblongata
- Respon pH rendah **[H+]>>**
  - Dalam cairan serebrospinal
- Stimulus **CO2** tinggi
  - Dalam cairan serebrospinal

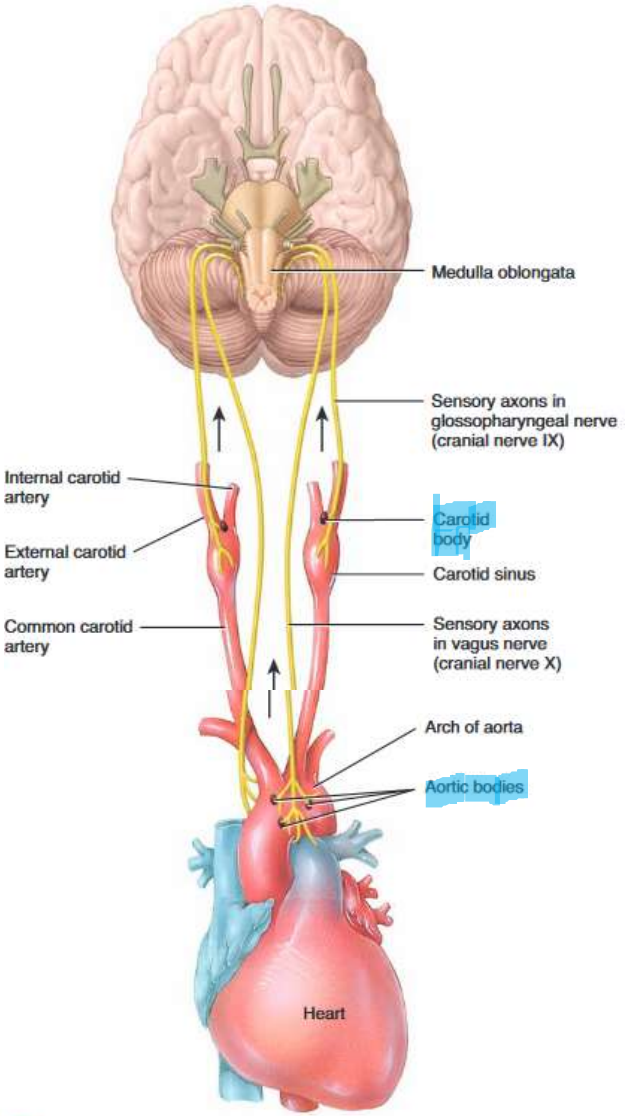
Carotid dan aortic bodies

- Lokasi dekat bifurkasio arteri karotis interna dan eksterna dan pada arkus aorta
- Stimulus **O2** rendah (masih diatas 50 mmHg)
- Dan stimulus **CO2** tinggi

Meningkatkan ventilasi alveolar

Figure 23.26 Locations of peripheral chemoreceptors.

Chemoreceptors are sensory neurons that respond to changes in the levels of certain chemicals in the body.

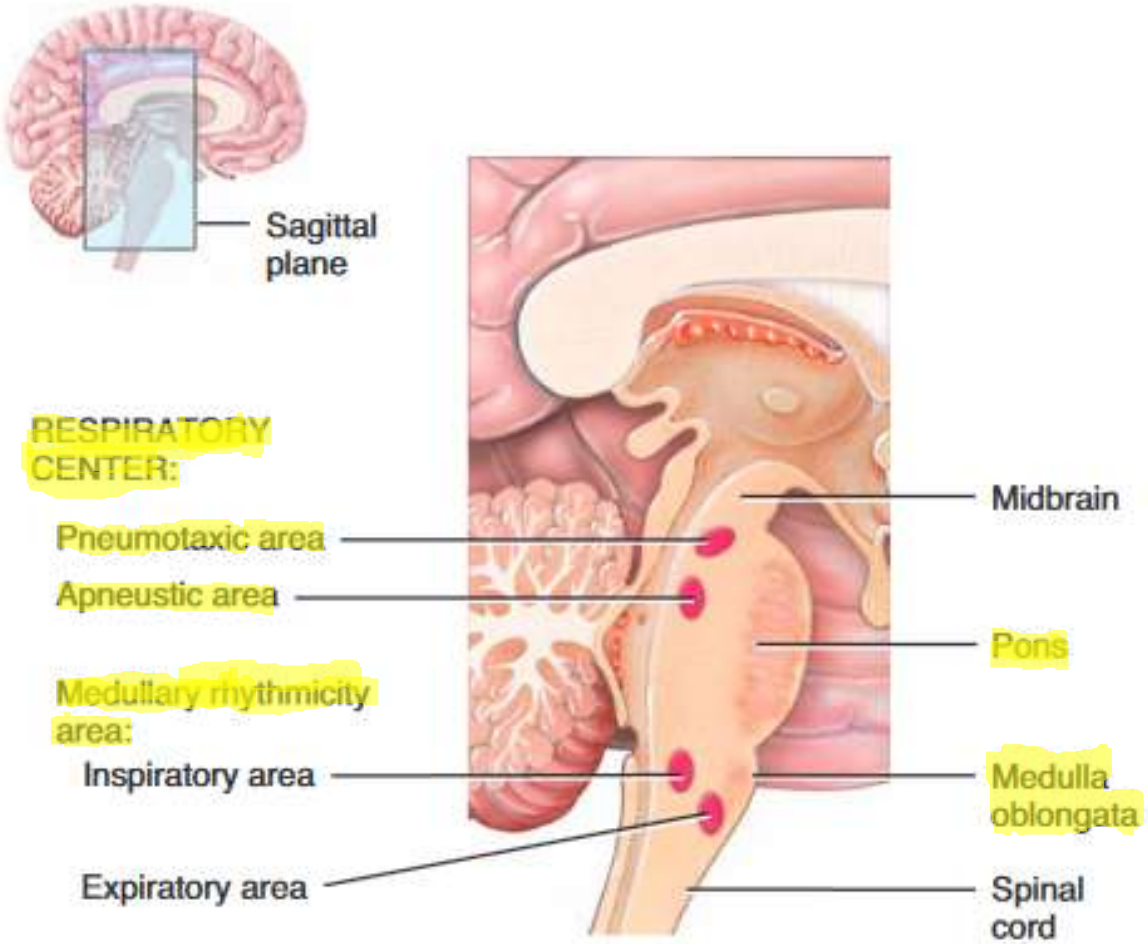


Which chemicals stimulate peripheral chemoreceptors?

Altitude (ft/meters)	Barometric Pressure (mm Hg)	Po_2 in Air (mm Hg)	*Pco_2 in Alveoli (mm Hg)*	Breathing A *Po_2 in Alveoli (mm Hg)*
0	760	159	40 (40)	104 (104)
10,000/3048	523	110	36 (23)	67 (77)
20,000/6096	349	73	24 (10)	40 (53)
30,000/9144	226	47	24 (7)	18 (30)
40,000/12,192	141	29		
50,000/15,240	87	18		

Figure 23.24 Locations of areas of the respiratory center.

The respiratory center is composed of neurons in the medullary rhythmicity area in the medulla oblongata plus the pneumotaxic and apneustic areas in the pons.

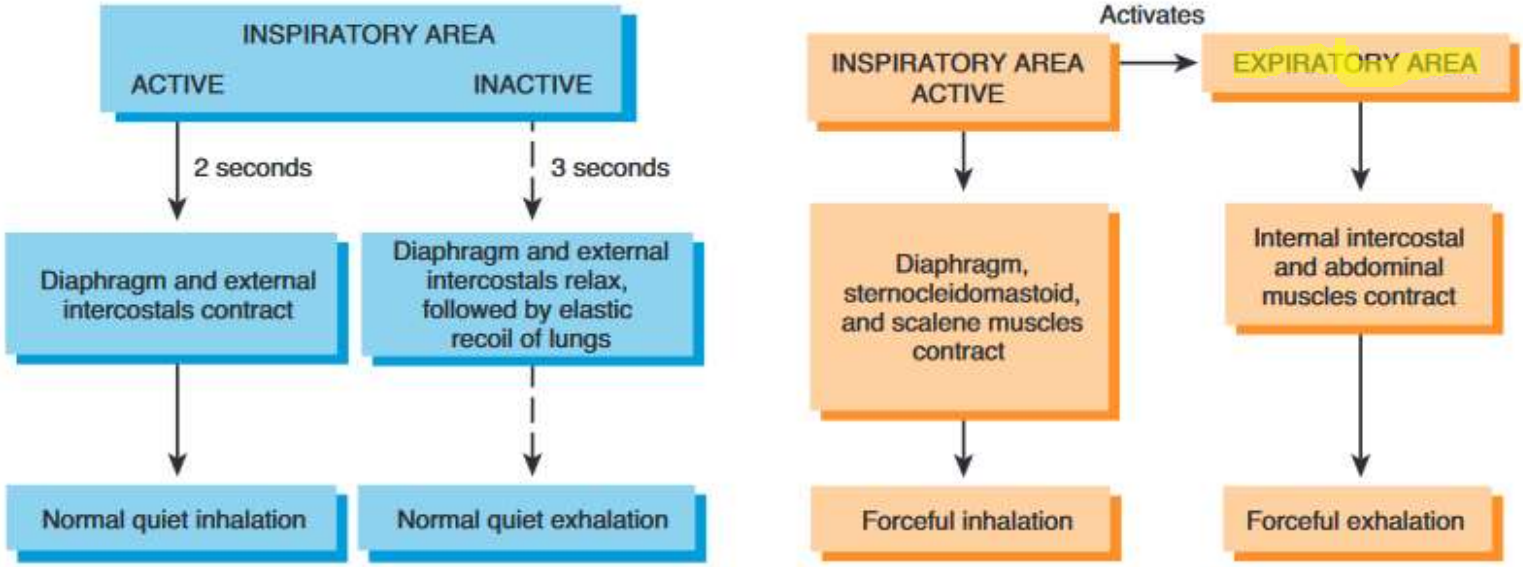


Sagittal section of brain stem

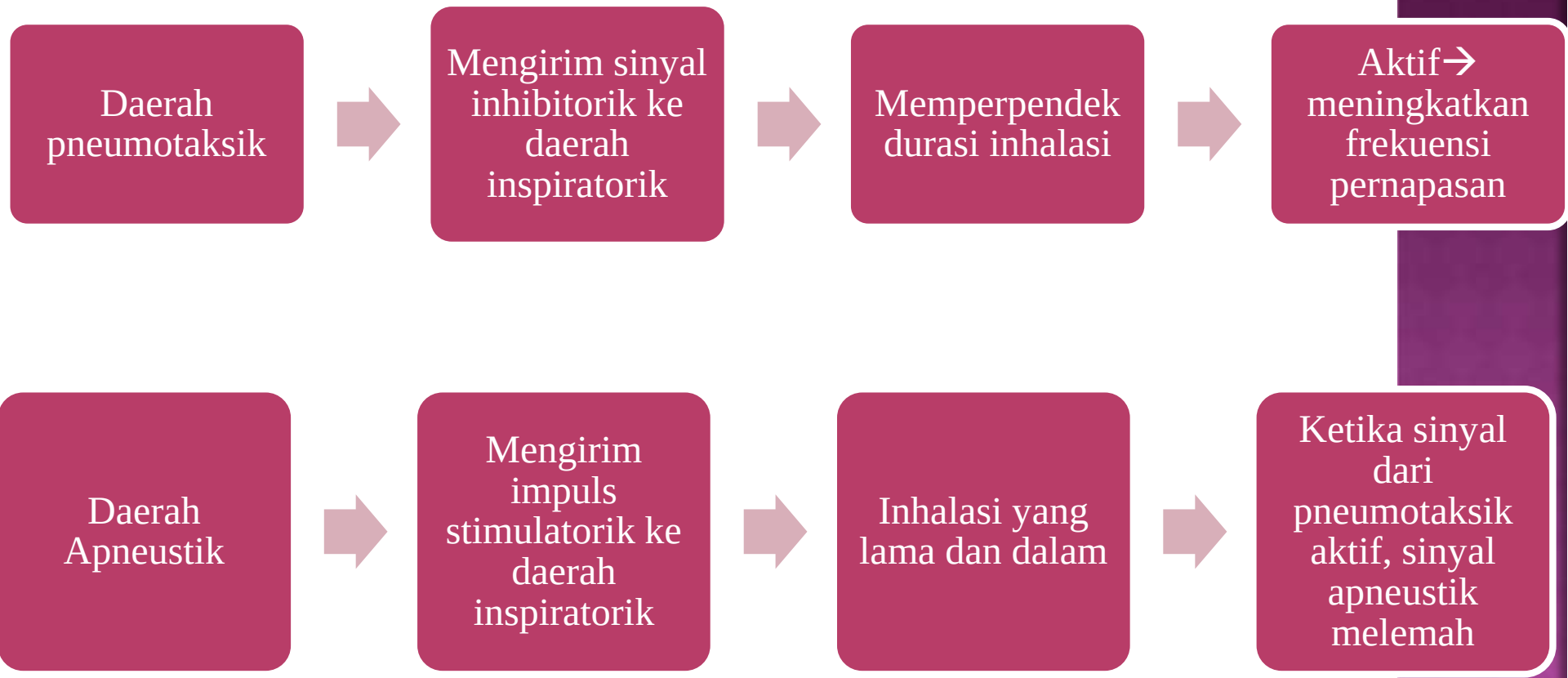
PERAN DAERAH RITMISITAS MEDULLA DALAM MENGONTROL IRAMA DASAR PERNAPASAN DAN PERNAPASAN KUAT

Figure 23.25 **Roles of the medullary rhythmicity area in controlling (a) the basic rhythm of respiration and (b) forceful breathing.**

During normal quiet breathing, the expiratory area is inactive; during forceful breathing, the inspiratory area activates the expiratory area.

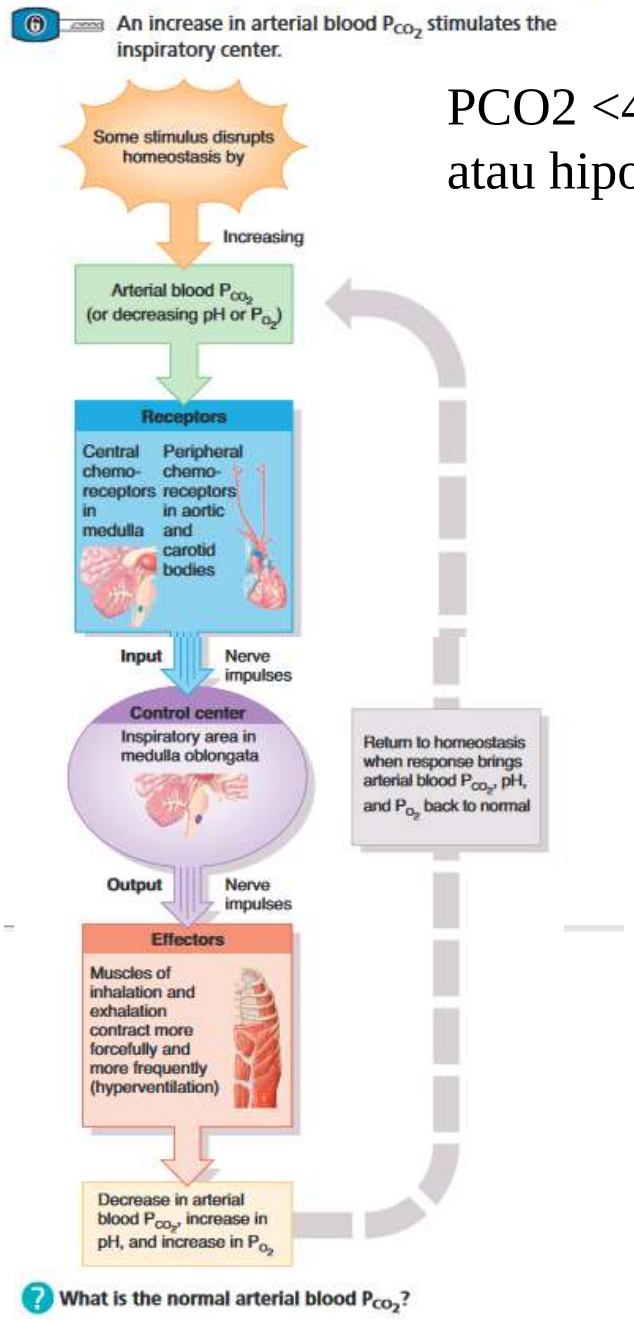


Which nerves convey impulses from the respiratory center to the diaphragm?


Daerah pneumotaksik
Mengirim sinyal inhibitorik ke daerah inspiratorik
Memperpendek durasi inhalasi
Aktif→ meningkatkan frekuensi pernapasan
Daerah Apneustik
Mengirim impuls stimulatorik ke daerah inspiratorik
Inhalasi yang lama dan dalam
Ketika sinyal dari pneumotaksik aktif, sinyal apneustik melemah

UMPAN BALIK NEGATIVE YG MENGATUR CO2, O2 DAN H+

Figure 23.27 Regulation of breathing in response to changes in blood P_{CO_2}, P_{O_2}, and pH (H^+) via negative feedback control.



PCO2 <40 mmHg→ hipokapnia atau hipokarbia→ kemoreseptor sentral

UMPAN BALIK POSITIF DGN KEMUNGKINAN AKIBAT YG MEMATIKAN

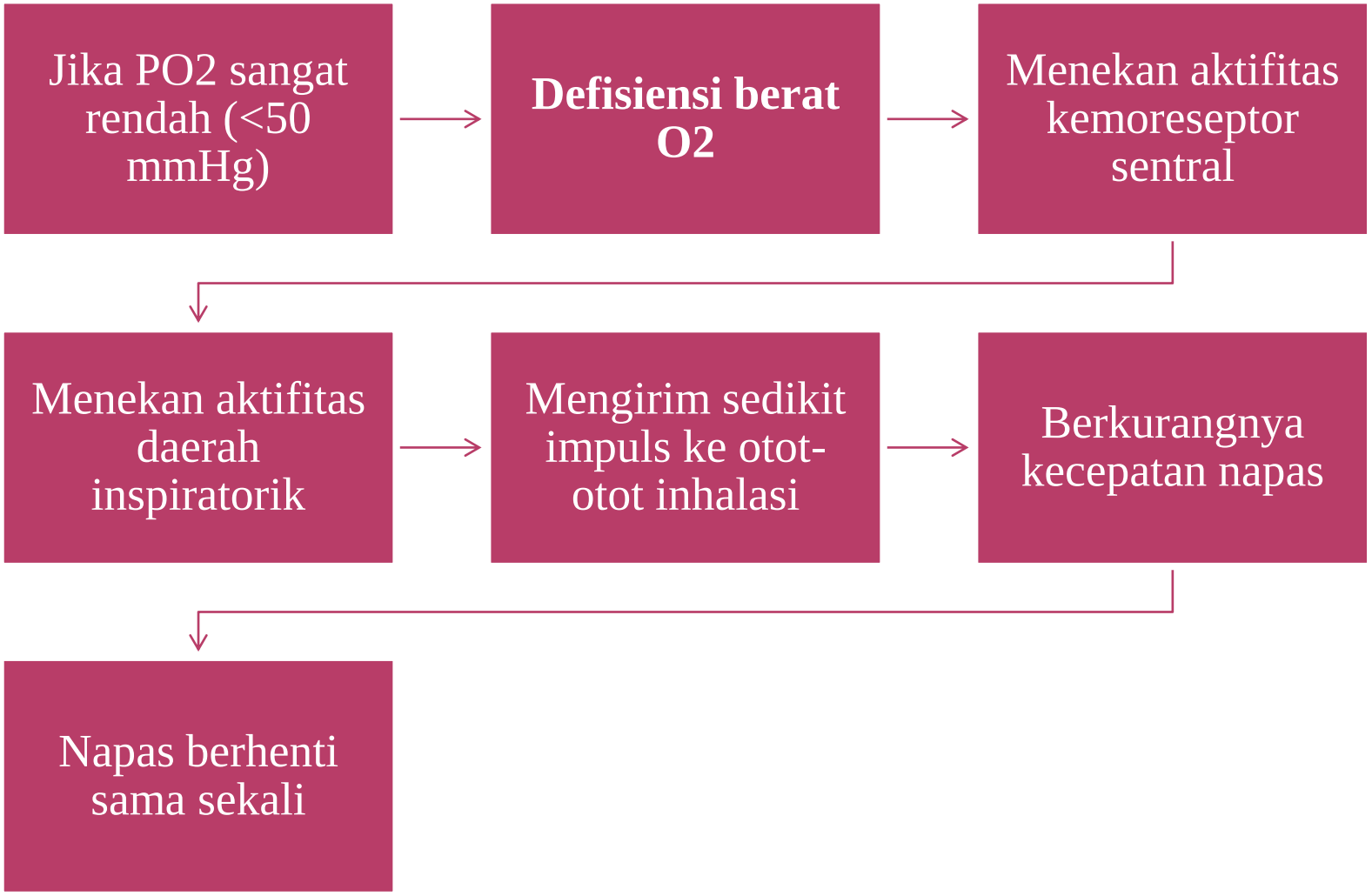


TABLE 23.3

Summary of Stimuli That Affect Ventilation Rate and Depth

STIMULI THAT INCREASE VENTILATION RATE AND DEPTH	STIMULI THAT DECREASE VENTILATION RATE AND DEPTH
Voluntary hyperventilation controlled by cerebral cortex and anticipation of activity by stimulation of limbic system.	Voluntary hypoventilation controlled by cerebral cortex.
Increase in arterial blood P_{CO_2} above 40 mmHg (causes an increase in H^+) detected by peripheral and central chemoreceptors.	Decrease in arterial blood P_{CO_2} below 40 mmHg (causes a decrease in H^+) detected by peripheral and central chemoreceptors.
Decrease in arterial blood P_{O_2} from 105 mmHg to 50 mmHg.	Decrease in arterial blood P_{O_2} below 50 mmHg.
Increased activity of proprioceptors.	Decreased activity of proprioceptors.
Increase in body temperature.	Decrease in body temperature (decreases respiration rate), sudden cold stimulus (causes apnea).
Prolonged pain.	Severe pain (causes apnea).
Decrease in blood pressure.	Increase in blood pressure.
Stretching of anal sphincter.	Irritation of pharynx or larynx by touch or chemicals (causes brief apnea followed by coughing or sneezing).

PENINGKATAN VENTILASI PARU-PERAN KEMORESEPTOR- HYPOXIC VENTILATORY RESPONSE (HVR)

Dalam beberapa menit setelah pendakian ke ketinggian **di atas 1500 m (4921 kaki)→ PO2** turun mendadak (**akut**)

↓

Merangsang kemoreseptor arteri (badan karotis)

↓

Meningkatkan ventilasi 1,65-5x normal

↓

PCO2 turun

↓

pH cairan tubuh meningkat ([H+] menurun)→ alkalosis respiratorik

↓

Menghambat pusat pernapasan (medula oblongata)→

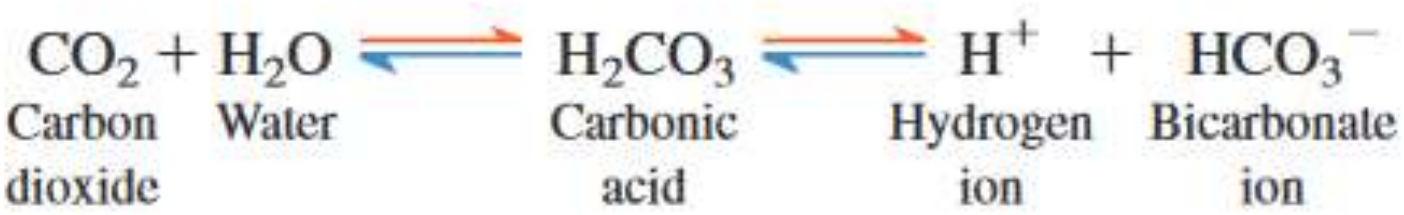
Melawan efek/stimulus O2 rendah pada badan karotid dan badan aortik

↓

Efek hambatan perlahan menghilang dlm waktu 2-5 hari→ rangsangan hipoksia kemoreseptor perifer dpt merangsang respon max kemoreseptor pusat

EKSHALASI KARBON DIOKSIDA

- Peningkatan 2x lipat ventilasi→ meningkatkan pH dari 7,4 menjadi 7, 63.
- Perlambatan ventilasi→ menurunkan pH dari 7,4 menjadi 7,00.



BAGAIMANA PENGARUH EFEK HAMBATAN PUSAT PERNAPASAN MEDULLA PERLAHAN HILANG (2-5 HARI)?

Penyebab→ penurunan kadar ion bikarbonat HCO3 dalam cairan serebrospinal.

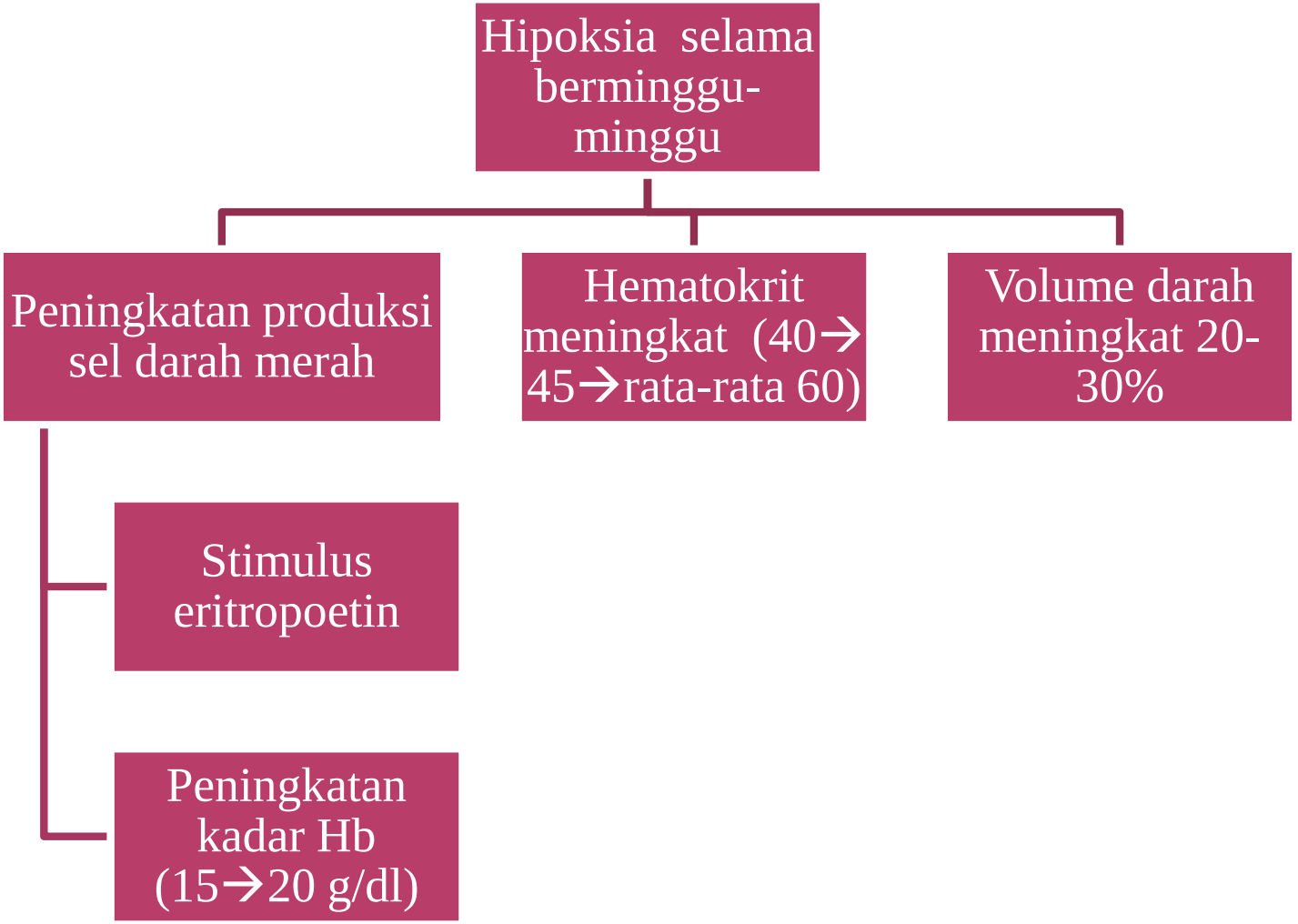
Kompensasi ginjal terhadap alkalosis respiratorik

- Stimulus PCO2 rendah ([H+] menurun →alkalosis respiratorik)
- Menurunkan sekresi ion hydrogen [H+]
- Meningkatkan ekskresi ion bikarbonat HCO3

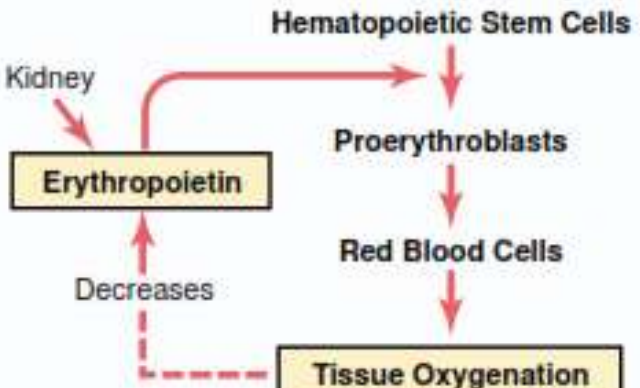
1. Konsentrasi bikarbonat menurun dlm cairan plasma dan serebrospinal
2. Menurunkan pH ke arah normal
3. Membuang efek inhibisi pernapasan akibat konsentrasi hidrogen rendah (alkalosis respiratorik)

2. PENINGKATAN JUMLAH SEL DARAH MERAH

PENINGKATAN JUMLAH SEL DARAH MERAH DAN KONSENTRASI HEMOGLOBIN SELAMA AKLIMATISASI

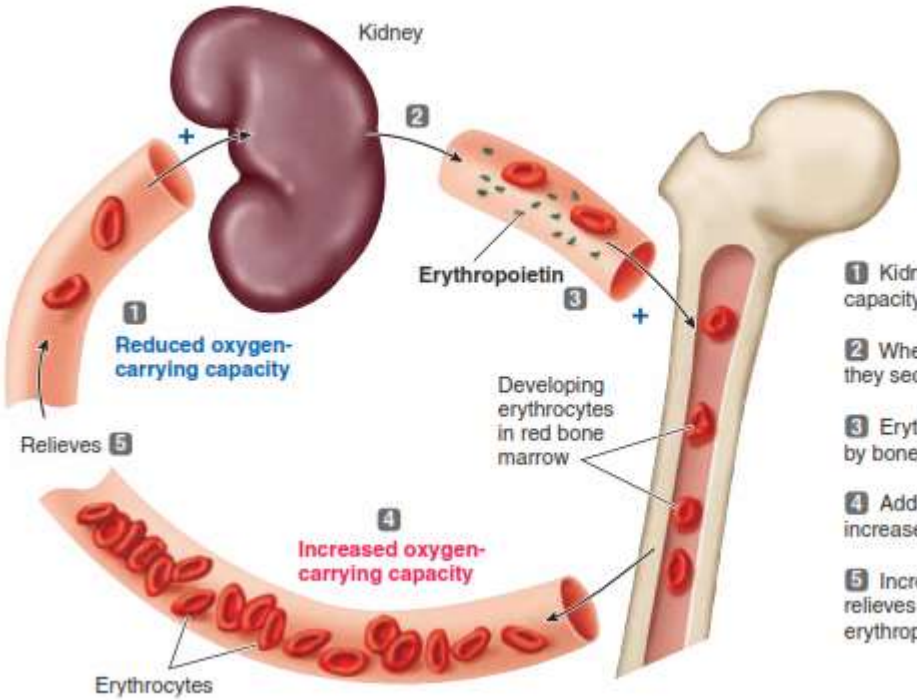


PENGATURAN PRODUKSI SEL DARAH MERAH - PERAN ERITROPOIETIN



Factors that decrease oxygenation

1. Low blood volu
2. Anemia
3. Low hemoglobi
4. Poor blood flow
5. Pulmonary dise



1 Kidneys detect reduced O_2-carrying capacity of blood.

2 When less O_2 is delivered to the kidneys, they secrete erythropoietin into blood.

3 Erythropoietin stimulates erythropoiesis by bone marrow.

4 Additional circulating erythrocytes increase O_2-carrying capacity of blood.

5 Increased O_2-carrying capacity relieves initial stimulus that triggered erythropoietin secretion.

3. PENINGKATAN KAPASITAS DIFUSI PARU

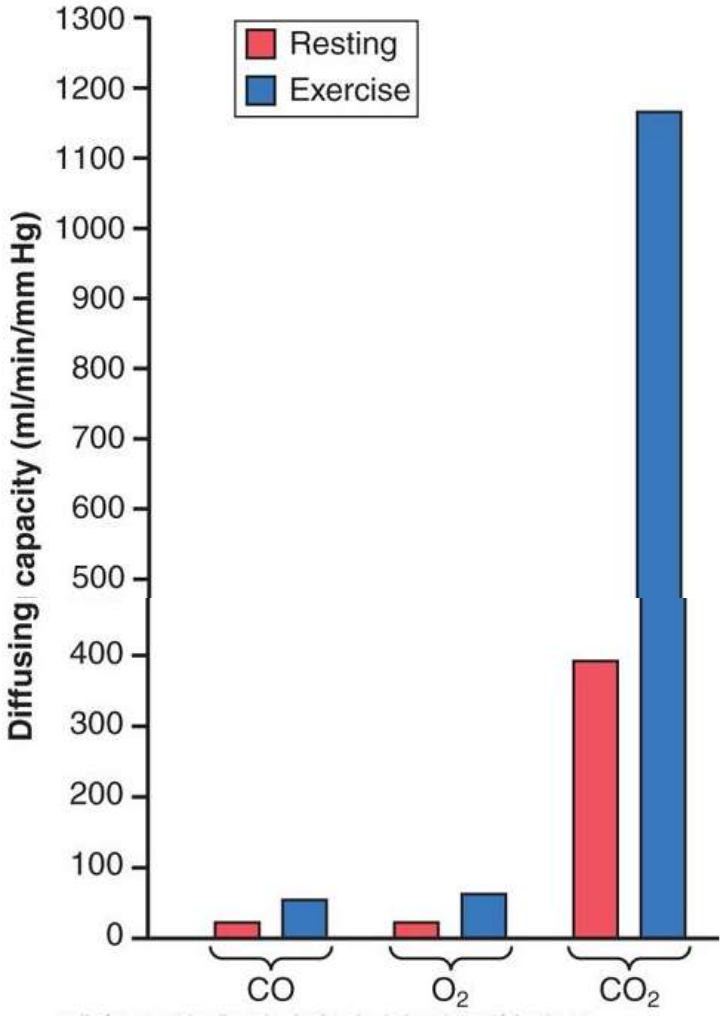
PENINGKATAN KAPASITAS DIFUSI PARU SETELAH AKLIMATISASI

- Kemampuan membran pernapasan dlm pertukaran gas antara alveoli dan darah paru dpt dinyatakan secara kuantitatif dgn **kapasitas difusi membran pernapasan**→ volume gas yg berdifusi melalui membran tiap menit pada setiap perbedaan tekanan parsial 1 mm Hg.

- Kapasitas difusi pada rata-rata lelaki dewasa muda dlm keadaan istirahat 21 ml/menit/mm Hg.


Vol. Darah Kapiler paru ↗
Vol. Paru ↗
Tek. Arteri paru ↗
Luas permukaan difusi ↗
Permukaan membran Alveolus ↗
Dorongan darah kapiler paru ↗
KAPASITAS DIFUSI ↗

KAPASITAS DIFUSI UNTUK KARBON MONOKSIDA, OKSIGEN DAN KARBON DIOKSIDA PADA PARU NORMAL PADA SAAT ISTIRAHAT DAN SELAMA KERJA FISIK



Faktor peningkatan kapasitas difusi:

- Pembukaan kapiler paru yg awalnya tdk aktif atau dilatasi ekstra pd kapiler yg telah terbuka.
- Pertukaran yg lebih baik ventilasi alveoli dan perfusi kapiler alveolus dgn darah (rasio ventilasi-perfusi)
- Peningkatan tekanan darah arteri paru mendorong darah untuk melalui banyak kapiler alveolus → bagian atas paru

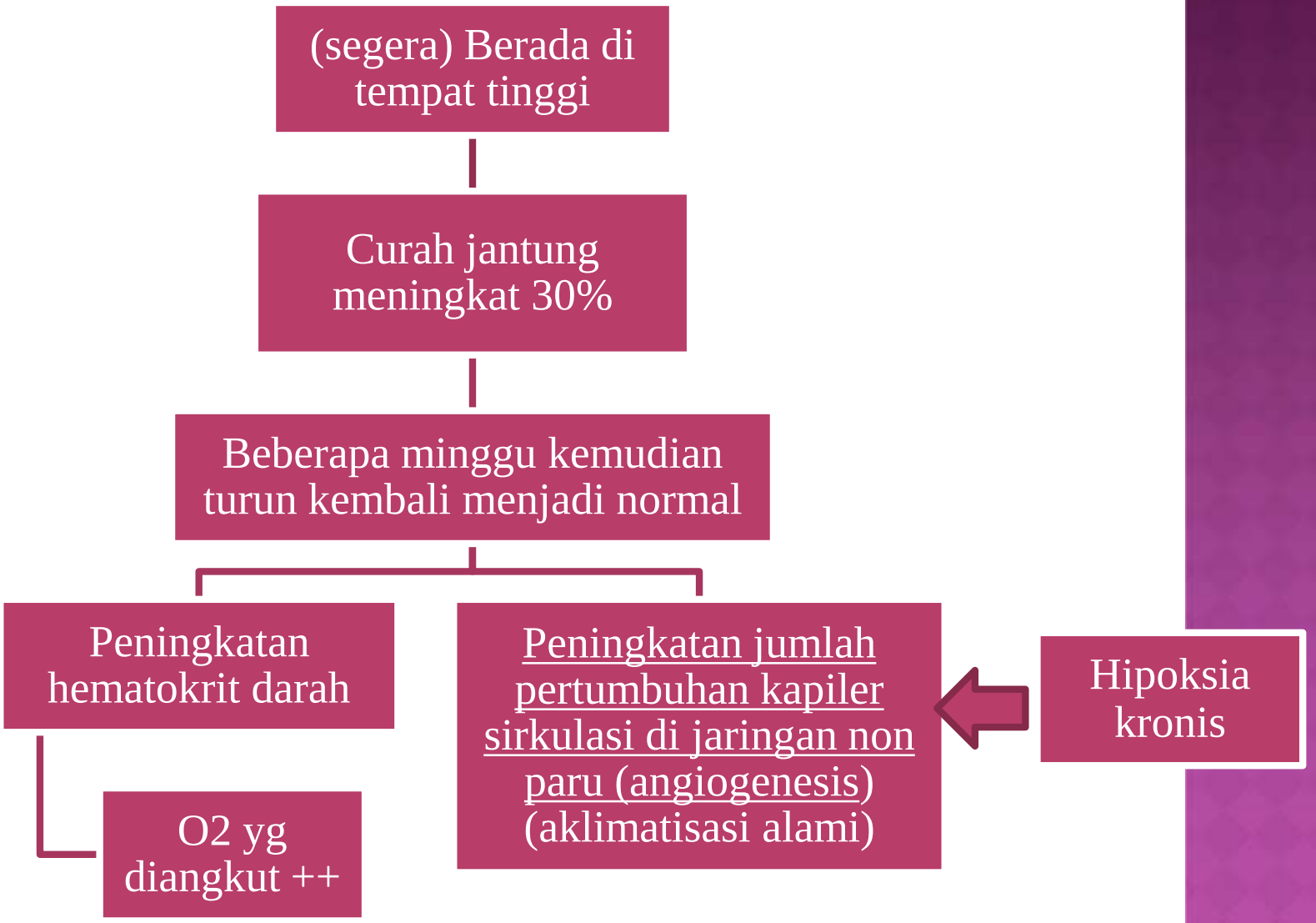
<u>Peningkatan kapasitas difusi juga terjadi di tempat tinggi.</u>

4. PENINGKATAN VASKULARISASI JARINGAN PERIFER

Cardiovascular adjustments

Cardiovascular adjustments of acclimatization include an initial increase in cardiac output and pulmonary perfusion, and selective vasoconstriction and vasodilation to help optimize oxygen uptake in the lungs and sustain oxygen delivery to the brain, heart, and other vital tissues [3].

PERUBAHAN SISTEM SIRKULASI PERIFER SELAMA AKLIMATISASI-PENINGKATAN KAPILARITAS JARINGAN



5. PENINGKATAN KEMAMPUAN SEL DALAM MENGGUNAKAN OKSIGEN SEKALIPUN NILAI PO2 RENDAH

AKLIMATISASI SEL

Binatang secara alami hidup di ketinggian 13.000-17.000 kaki

Sistem mitokondria sel dan enzim oksidatif sel sedikit lbh banyak

Dapat menggunakan oksigen lebih efektif

AKLIMATISASI ALAMI PADA PENDUDUK ASLI YANG HIDUP DI TEMPAT TINGGAL

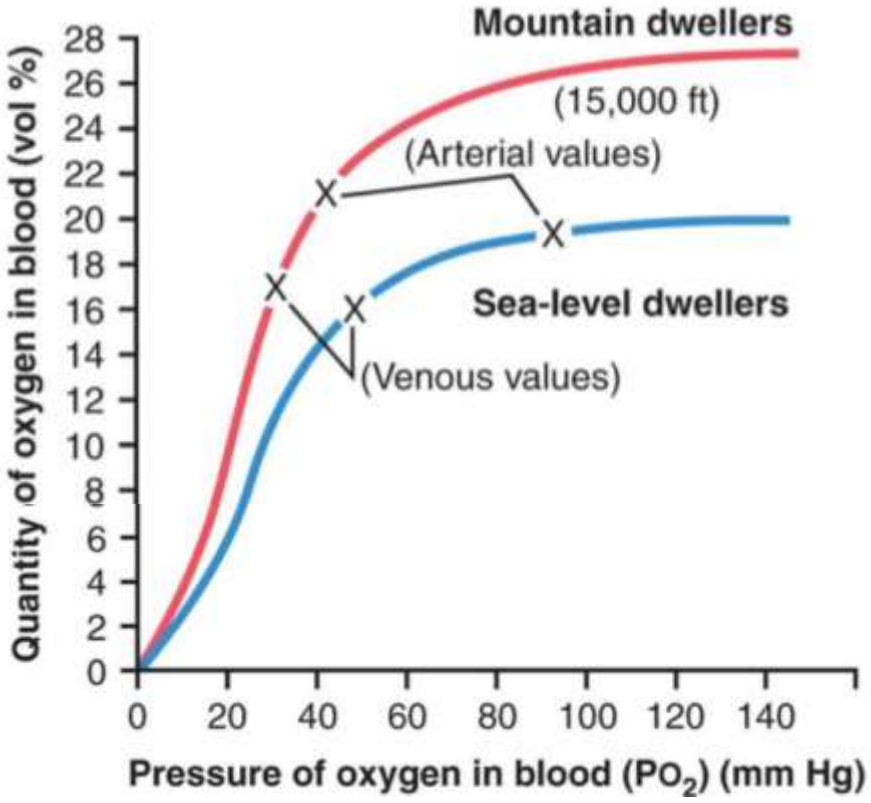
Penduduk asli pegunungan Andes dan Himalaya pada ketinggian >13.000 kaki

Proses aklimatisasi semenjak masa bayi

Ukuran dadanya sangat besar, ukuran tubuhnya agak mengecil→rasio kapasitas ventilasi terhadap massa tubuh menjadi tinggi

Sejak lahir hingga dewasa sudah memompa curah jantung dlm jumlah ekstra dan memiliki ukuran yg lebih besar

KURVA DISOSIASI OKSIGEN PADA PENDUDUK YANG TINGGAL DI TEMPAT TINGGI DAN TEMPAT SETINGGI PERMUKAAN LAUT



- Penduduk yang tinggal di tempat setinggi 15.000 kaki

PENURUNAN KAPASITAS KERJA DI TEMPAT TINGGI DAN EFEK POSITIF AKLIMATISASI

In general, work capacity is reduced in direct proportion to the decrease in maximum rate of oxygen uptake that the body can achieve.

	Work capacity (percent of normal)
Unacclimatized	50
Acclimatized for 2 months	68
Native living at 13,200 feet but working at 17,000 feet	87

To give an idea of the importance of acclimatization in increasing work capacity, consider the large differences in work capacities as percent of normal for unacclimatized and acclimatized people at an altitude of 17,000 feet:

- Kapasitas kerja semua otot sangat menurun pada hipoksia
- Kapasitas kerja turun sebanding dengan ambilan oksigen maksimal yang dapat dicapai oleh tubuh
- **Pentingnya aklimatisasi** dalam meningkatkan kapasitas kerja.

MOUNTAIN SICKNESS

ACUTE mountain sickness is a symptom-complex; that is, a *group* of symptoms usually appearing together, with variations among individuals. It is seen in people who have recently arrived at high altitude or have just gained altitude, and may start anytime from a few hours to a few days after ascending. *High altitude* is arbitrarily defined as altitudes greater than 3,000 meters (10,000 feet). People with heart and lung diseases may have difficulty at lower altitudes and, rarely, a healthy person will develop some form of acute mountain sickness as low as 2,500 meters (8,200 feet). Most commonly acute mountain sickness is seen above 3,000 meters (10,000 feet).

Hackett Peter H. 1993. Mountain sickness. The American Alpine Club

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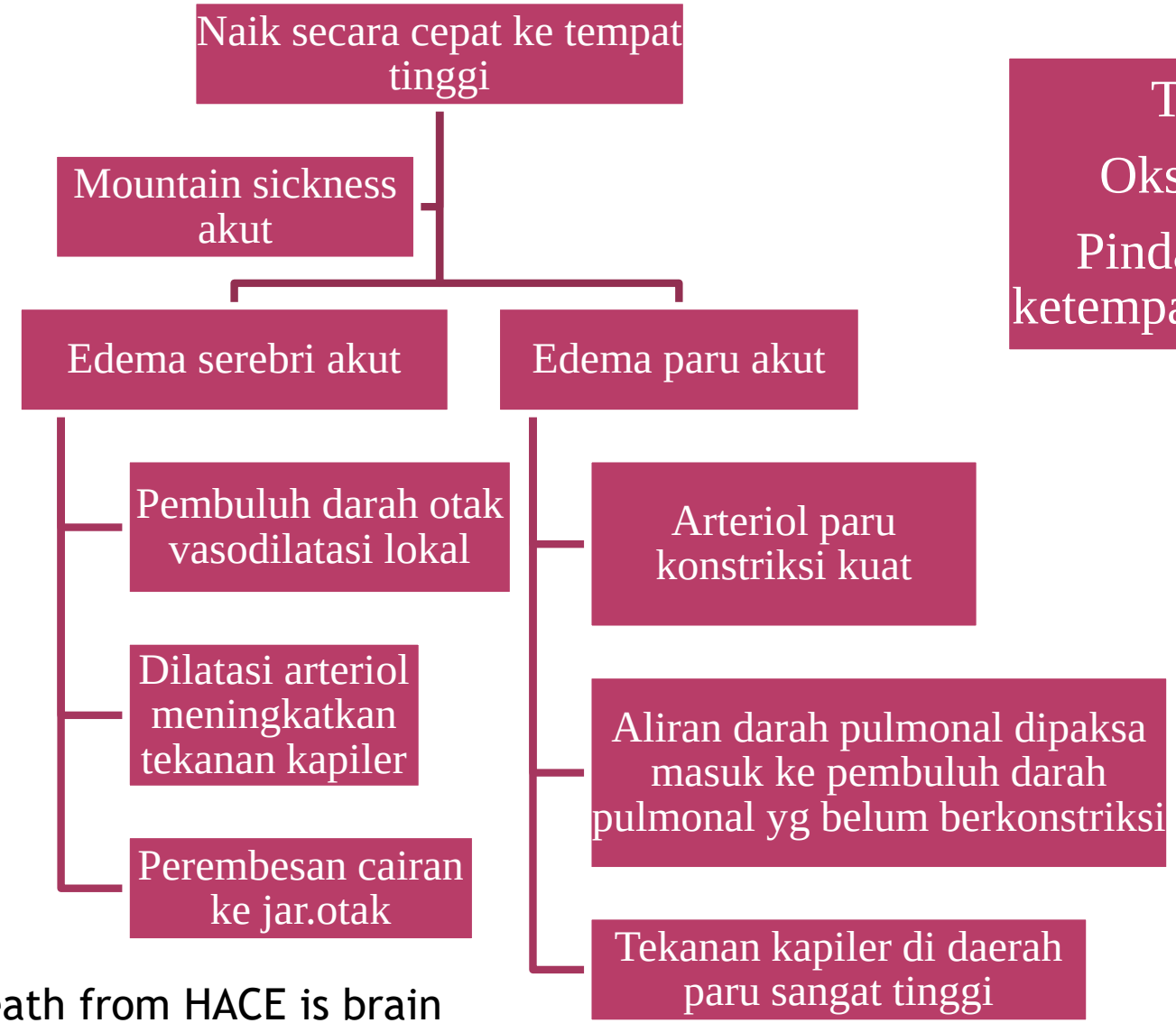
MOUNTAIN SICKNESS AKUT DAN EDEMA PARU AKUT PADA KETINGGIAN



Tx:

Oksigen

Pindahkan ketempat rendah

The cause of death from HACE is brain herniation from severely increased intracerebral pressure.

- Sifat unik pembuluh darah paru→ konstriksinya sebagai respon terhadap **hipoksia** local. Di semua jaringan tubuh lain, hipoksia menyebabkan dilatasi pembuluh darah untuk meningkatkan aliran darah.

- Vasokonstriksi sebagai respon terhadap hipoksia akan mengalihkan darah paru dari bagian paru yg kurang mendapat ventilasi ke bagian yg berventilasi baik agar pertukaran gas menjadi lebih efesien→ fenomena pemandu ventilasi-perfusi.

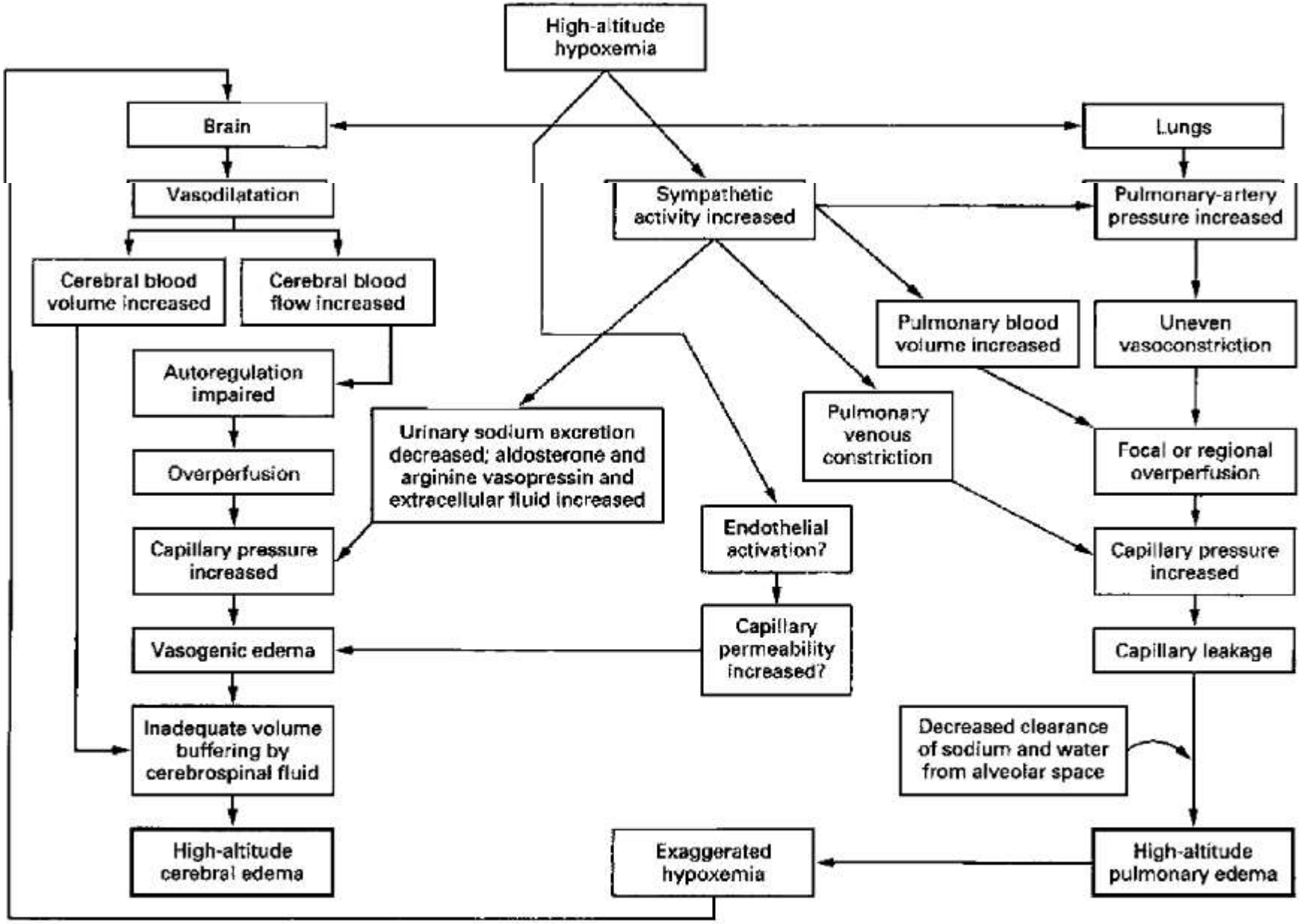


Fig. 2. Proposed pathogenesis of high-altitude illness. (*From* Hackett P, Roach RC. High-altitude illness. N Engl J Med 2001;345:107–14; with permission.)

DIAGNOSIS AMS

- Diagnosis AMS bersifat klinis; tidak ada modalitas diagnostik atau temuan fisik yang handal konfirmasikan diagnosisnya.
- The Lake Louise Consensus Committee: AMS adalah sakit kepala dan satu atau lebih dari berikut ini: anoreksia, mual, atau muntah; kelelahan atau kelemahan; pusing; atau kesulitan tidur.
- Gejala ini harus terjadi pada saat kedatangan baru-baru ini sampai di tempat ketinggian.

HEADACHE
INSOMNIA
LASSITUDE
LOSS OF COORDINATION (ATAXIA)
EDEMA OF THE EYES AND FACE

COUGH
SHORTNESS OF BREATH
FULLNESS OR TIGHTNESS IN CHEST
IRREGULAR BREATHING, ESPECIALLY AT NIGHT

LOSS OF APPETITE
NAUSEA
VOMITING

REDUCED URINE OUTPUT

WEAKNESS

LEGS FEEL "HEAVY"

TINDAKAN PREVENTIF TERHADAP AMS

- Mencoba dengan kenaikan bertingkat (istirahat setiap 600-1200 m) dan laju pendakian yang lambat (maksimum 600 m/hari) memungkinkan waktu yang cukup untuk aklimatisasi dan mengurangi risiko semua jenis penyakit ketinggian tinggi, termasuk AMS.

- Namun demikian, tidak ada cara yang dapat diandalkan untuk memprediksi risiko AMS

INSIDEN AMS

- Insiden dan tingkat keparahan penyakit ketinggian akan meningkat diiringi dgn bertambahnya ketinggian dan tingkat pendakian, karena masing-masing faktor mempengaruhi besarnya stres hipoksia.

- Tingkat kejadian AMS pada populasi wisatawan umum sekitar 2500 m (8202 kaki) dilaporkan mencapai 20% sampai 25%. Meningkat menjadi 40% sampai 50% di trekker dan pendaki dekat 4000 m (13.123 kaki).

- Bila pendakian sekitar 4000 m terjadi selama beberapa jam sampai hari, kejadian AMS meningkat menjadi lebih dari 90%.

INSIDEN HACE

Tingkat kejadian HACE pada populasi umum sekitar 2500 m (8202 kaki) umumnya dilaporkan kurang dari 0,01% namun meningkat menjadi 1% sampai 2% di trekker, pendaki, dan tentara di dekat 4000 m

DIAGNOSIS HACE

- Bersifat klinis;
- Ciri kardinal adalah perubahan dalam kesadaran dan ataksia
  Perubahan status mental termasuk perilaku irasional yang cepat berkembang menjadi kelesuan, obtundasi, dan koma.
- Ataksia truncal adalah diagnostic tanda fisik; papilledema, perdarahan retina, kelumpuhan saraf kranial, Refleks abnormal dan defisit neurologis fokal juga mungkin ada.
- Kematian akibat herniasi otak

INSIDEN HAPE

- Tingkat kejadian HAPE juga meningkat dengan tingkat ketinggian dan pendakian, berkisar antara 0,01% sampai 0,1% pada populasi turis umum pada ketinggian 2.500 m (8202 kaki) sampai 2% sampai 6% di trekker dan pendaki dekat 4000 m (13.123 kaki).

- Saat pendakian ke 5500 m (18.044 kaki) terjadi selama beberapa jam sampai hari, kejadian HAPE meningkat dari 2,5% menjadi 15,5%.

MOUNTAIN SICKNESS KRONIS

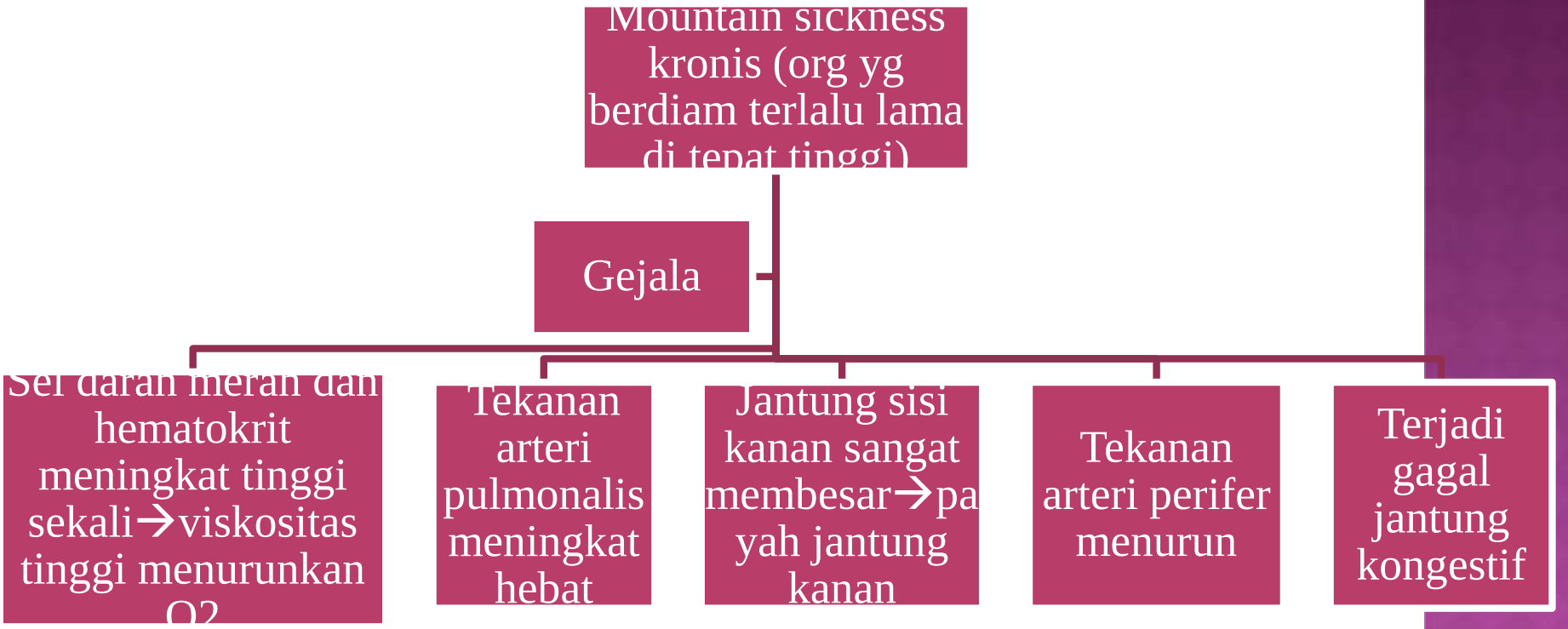


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Table 2
Management options for high-altitude illness

High-altitude illness	Treatment setting	
Type and severity	Wilderness	High-altitude hospital or clinic
Mild AMS Mild headache, nausea, decreased appetite, dizziness, fatigue, and poor sleep	Descent ≥500 m (1640 ft), or acclimatization for 1–2 days at the same altitude; avoid further ascent until symptoms resolved; consider acetazolamide (125–250 mg PO BID) to speed acclimatization; analgesics and antiemetics as needed	Treatment same as in field, but oxygen available; oxygen (1–2 L/min for 12–24 h) resolves symptoms and allows return to exertion or further ascent in 1–2 days; acetazolamide speeds acclimatization
Moderate AMS Protracted or worsening headache, nausea, vomiting, dizziness, anorexia, or fatigue	Descend, consider dexamethasone (4 mg PO/IM every 6 hours); and/or acetazolamide (125–250 mg PO BID); if unable to descend, vigilant observation for deterioration; oxygen (1–2 L/min) and/or portable hyperbaric therapy (2–4 psi) for a few hours if available	Outpatient oxygen (1–2 L/min continuously) for 1–2 days until symptoms resolve; consider one to two doses of dexamethasone 4 mg q 6 h, consider acetazolamide if remaining at altitude
HACE Mental status changes (confusion, bizarre behavior, severe lassitude), and ataxia	Immediate descent or evacuation ≥1000 m (3281 ft); oxygen to maintain SaO_2 ≥90%; dexamethasone (8 mg IV/IM/PO initially, then 4 mg q 6 h); HB therapy if cannot descend	Treatment same as in field; coma care as needed; consider brain MRI if diagnosis unclear; avoid cerebral ischemia from excessive hyperventilation or hypovolemia from diuretics

HAPE		
Dyspnea at rest, moist cough, rales, severe exercise limitation, cyanosis, tachypnea, tachycardia, desaturation, infiltrates on radiograph	Oxygen (4–6 L/min until improved, then 2–4 L/min to conserve supplies and maintain SaO_2 ≥90%); minimize exertion and keep patient warm; descend or evacuate ≥500–1000 m (1640–3281 ft) as soon as possible; if descent/O_2 unavailable, portable HB therapy (2–4 psi continuously) may be lifesaving; consider nifedipine (10 mg PO, then 30 mg extended-release PO q 12–24 h) if no HACE; consider inhaled beta-agonists (salmeterol, 125 μg inhaled q 12 h, or albuterol); consider EPAP mask; dexamethasone only if HACE develops	Descent provides quickest recovery, but rest and oxygen sufficient for all but the most severe cases and those complicated by HACE, as long as SaO_2 remains ≥90% with 2–4 L/min oxygen; start oxygen (4–6 L/min or more, titrated to SaO_2 ≥90%) under observation for several hours, followed by outpatient continuous oxygen (usually, 1–3 days at 1–4 L/min, titrated to SaO_2 ≥90%) with oxygen concentrator for home and portable unit for brief outings, until RA SaO_2 ≥90%, or descent; check RA SaO_2 q 12–24 h; admit those with HACE, oxygen requirement >4 L/min, co-morbid conditions, children, and elderly; beta-agonists, nifedipine and hyperbaric therapy probably of no additional benefit if SaO_2 ≥90% with oxygen

Abbreviations: HB, hyperbaric; IM intramuscular; IV, intravenous; RA, room air.

Table 3
Therapeutic options for high-altitude illness

Therapy	Use	Dose	Mechanism	Negative effects	Comments
Rest and halt ascent	Treatment for all high-altitude illness	Avoid strenuous activity; no further ascent	Permits acclimatization; avoids further hypoxemic stress from exertion and continued ascent	May be inconvenient, impractical, particularly if symptoms are mild	The principal treatment for mild to moderate AMS; mild illness may completely resolve in 1–2 days; always use for HAPE and HACE, if descent not possible
Descent	Treatment for all forms of high-altitude illness	300 m (984 ft) or more	Reverses hypobaric hypoxia by increasing PIO_2	May be impractical or impossible; inconvenient for mild illness	Continue descent until patient improved; for HACE and HAPE, do not delay to wait for help
Oxygen	Treatment for all forms of high-altitude illness	2–15 L/min to maintain $SaO_2 \geq 90\%$, until symptoms improve	Oxygen reverses hypoxia by raising FIO_2	Oxygen is bulky, heavy, and often unavailable or in limited quantity	When available always use for HAPE and HACE; mild AMS may improve with only 1–2 h of low-flow therapy; continuous therapy for HAPE until RA $SaO_2 \geq 90\%$ or patient descends
EPAP	Treatment of HAPE	Continuously until descent or oxygen available	Improves gas exchange and SaO_2	Increases work of breathing	May be useful temporizing measure when supplemental oxygen and descent not available

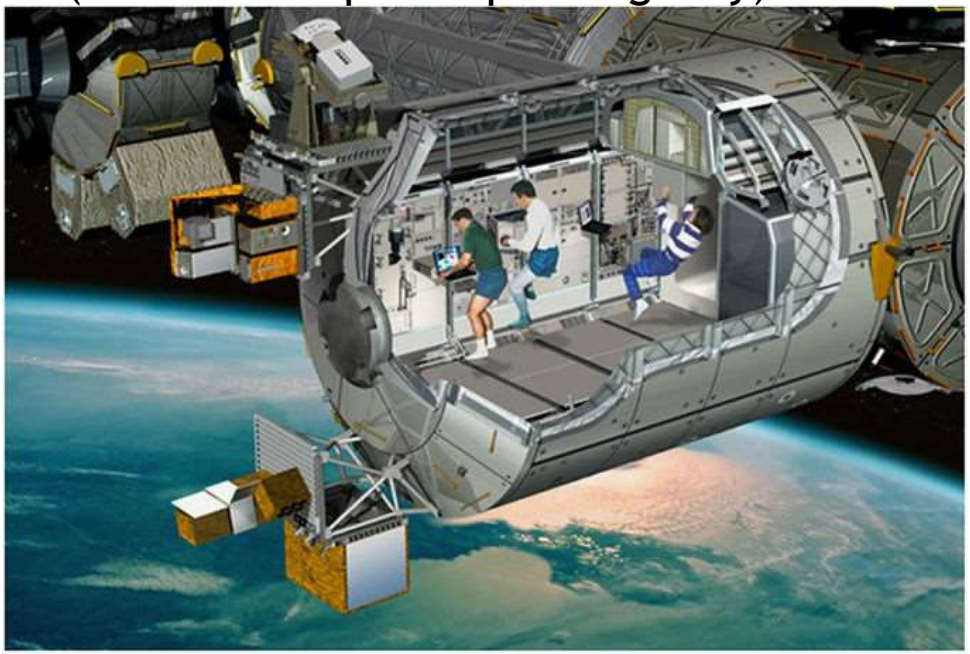
Hyperbarics: Gamow bag, Chamberlite [60,62,63]	Treatment for all high-altitude illness	2-15 psi until symptoms improve or descent possible; mild AMS may improve with only 1–2 h of therapy; HAPE and HACE require continuous therapy if O_2/descent not available	Simulated descent reverses hypoxia by increasing PIO_2; HB and O_2 therapy raise SaO_2 and improve symptoms comparably; Chamberlite durable up to 15 psi	Gamow bag requires continuous pumping to pressurize and ventilate; effects short lived; rebound possible; no proven advantage over oxygen	Alternative to bottled oxygen in remote places; valuable adjunct to other treatments because of rapid action; Gamow bag light and portable; Chamberlite far less portable but goes to 15 psi, ventilated by compressed gas (scuba tank)
Acetazolamide	Prevention of AMS	125–250 mg PO BID, starting day before ascent and continued for 2 days at maximum altitude	Promotes acclimatization by stimulating bicarbonate diuresis and respiration	Contraindicated in sulfonamide allergic; pregnancy class C; paresthesias; alters taste of carbonated beverages; polyuria	No rebound effect; may be combined with dexamethasone for treatment of AMS/ HACE
	Treatment of AMS	125–250 mg PO BID, until symptoms resolve; pediatric dose: 2.5 mg/kg BID			
Dexa-methasone [64–67]	Prevention of AMS	2 mg q 6 h, or 4 mg q 12 h PO, starting day of ascent and discontinued cautiously after 2 days at maximum altitude	Unknown, may work by reducing brain blood volume or blocking brain capillary leakage	Rebound possible when stopped, no effect on acclimatization; mood changes, hyperglycemia, dyspepsia, best avoided in pregnancy	Can be lifesaving for AMS or HACE; rapid onset in 2–8 h; more gradual but longer lasting relief than HB alone; no value in HAPE
	Treatment of AMS	4 mg q 6 h PO/IV/IM			
	Treatment of HACE	8 mg PO/IV/IM initially, then 4 mg q 6 h			

Table 3 (*continued*)

Therapy	Use	Dose	Mechanism	Negative effects	Comments
Nifedipine	Prevention of HAPE	20–30 mg extended-release BID PO, start day prior to ascent and continue for 2 days at maximum altitude	Reduces PA pressure by acting on smooth musculature	Hypotension and reflex tachycardia possible, but uncommon; pregnancy class C	No value in AMS/HACE; not necessary if oxygen available
	Treatment of HAPE	10 mg, then 20–30 mg extended release BID			
Beta-agonists; salmeterol, albuterol	Prevention of HAPE [19]; possible treatment of HAPE	125 μg inhaled BID, starting day before ascent until 2 days at maximum altitude; optimal dose of albuterol unknown	Upregulate active alveolar transepithelial sodium transport, increase clearance of alveolar fluid	Rare	Anectodal evidence suggests albuterol effective in the treatment of HAPE, but has yet to be studied; consider combining with nifedipine for treatment of HAPE if oxygen or descent not available
Grinko biloba [32]	Prevention of AMS	Unknown: 120–180 mg/day in divided doses, begin 1–5 days before ascent	Unknown, possible antioxidant effects	Occasional headache, rarely bleeding	Rare side effects; preparations vary; safe in pregnancy

Abbreviations: EPAP, expiratory positive airway pressure; HB, hyperbaric; IM, intramuscular injection; IV, intravenous injection; RA, room air.

Columbus module of the ISS (Credit: European Space Agency).



SPACE SICKNESS

Space sickness, space adaptation syndrome, space motion sickness. All labels for the contemporary motion sickness phenomenon experienced by space travellers.
The first recorded episode of space sickness was from the second human to orbit Earth, Gherman Titov in 1961

EFEK GAYA PERCEPATAN TERHADAP TUBUH PADA FISIOLOGI PENERBANGAN DAN LUAR ANGKASA

Beberapa jenis gaya percepatan memengaruhi tubuh sewaktu kita terbang.

GAYA PERCEPATAN SENTRIFUGAL

- Ketika sebuah pesawat terbang berbelok, gaya percepatan sentrifugal yg ditimbulkan dapat dihitung dgn persamaan berikut:

$$f = \frac{mv^2}{r}$$

f = gaya percepatan sentrifugal

m = massa benda

v = kecepatan terbang

r = jari-jari kelengkungan belokkan

Gaya percepatan sentrifugal akan meningkat sebanding dengan kuadrat kecepatan. Gaya percepatan benbanding lurus dengan ketajaman belokkan.

PENGUKURAN GAYA PERCEPATAN-"G"

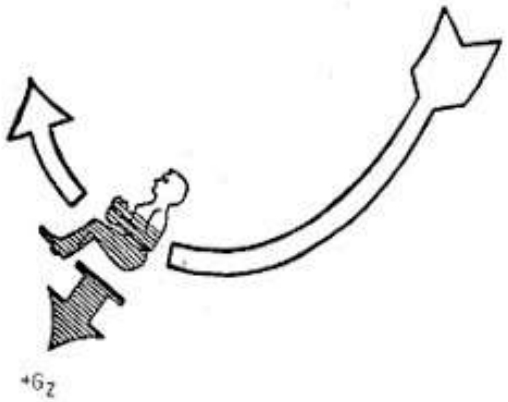
G positif

Sistem sirkulasi

- Darah akan tertarik ke bag. terbawah tubuh

Vertebrata

- +20G batas max dpt ditahan o/manusia dalam posisi duduk



- Bila +5G dlm posisi berdiri diam→ tekanan vena kaki meningkat (450 mm Hg)
- Bila +5G dlm posisi duduk→tekanan vena 300 mm Hg

↓

Darah >> dari bag atas tubuh berpindah ke bawah (dilatasi pasif pembuluh darah bagian inferior)

↓

Jantung tdk dapat memompa secara efektif krn darah yg kembali ke jantung menurun

↓

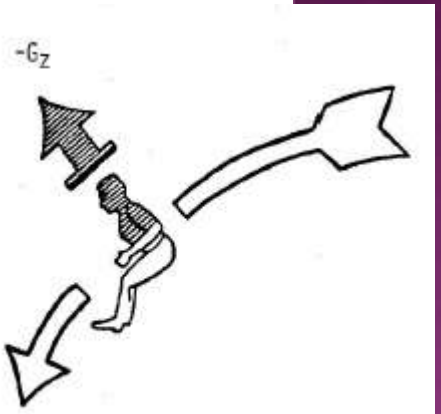
Curah jantung menurun

Batas teraman 10 G dgn duduk

G Negatif

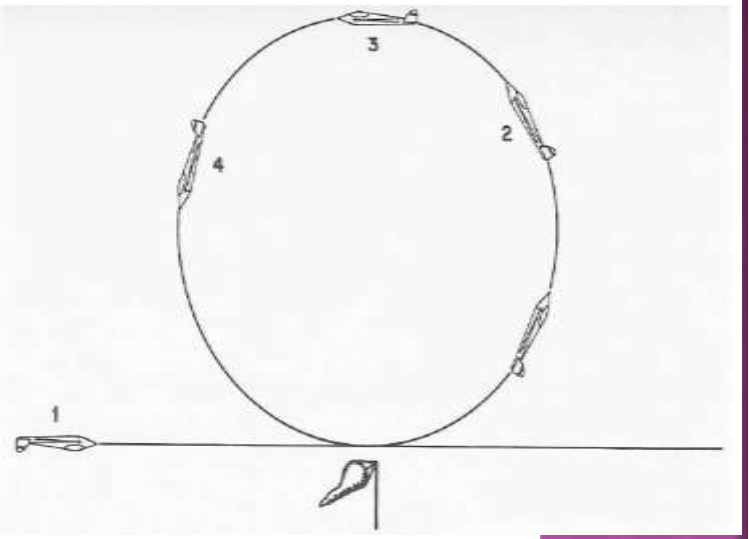
Pada manuver outside loop (-4 G s/d -5 G)

- Tanpa terkena efek buruk permanen (akut)
- Hyperemia berat sejenak
- Kadang Gangguan psikotik (15-20 mnt)→edema otak



Batas max -20G

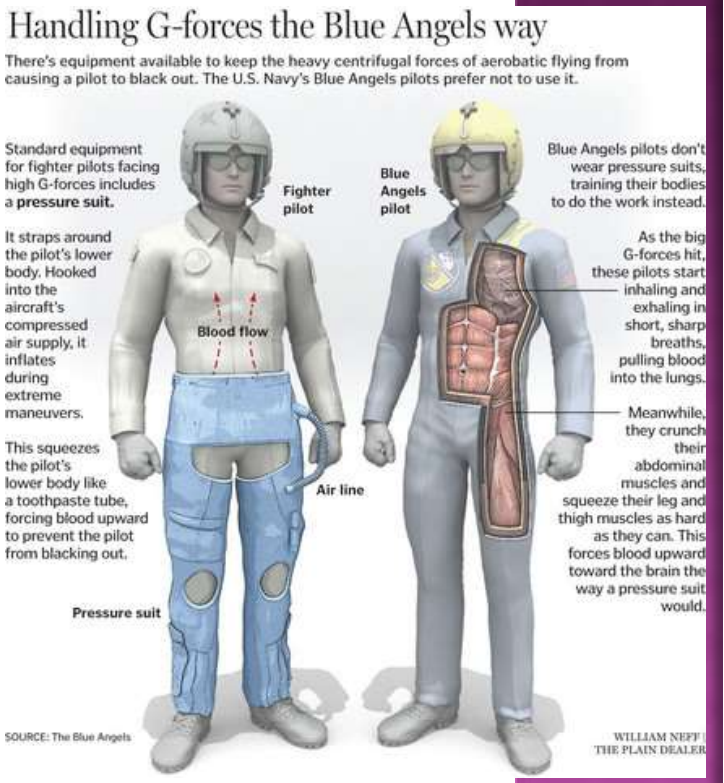
- Sentifugasi darah ke kepala tinggi
- Meningkatkan tekanan pembuluh otak mnjd 300-400 mm Hg
- Buta mata sementara



PERLINDUNGAN TUBUH TERHADAP GAYA PERCEPATAN SENTRIFUGAL

Menghindari kolaps sirkulasi pada G positif:

- Mengencangkan otot-otot perut dgn sangat kuat dan membungkuk ke depan sambil menekan perutnya→penimbunan darah di pembuluh-pembuluh darah besar abdomen dpt dihindari. "**blackout**"
- Memakai pakaian khusus anti G→ mencegah penimbunan darah di perut bag. Bawah dan tungkai.



Symptoms of exposure to high vertical G begin with tunnel vision, then loss of central vision (grey-out), then loss of vision altogether (black-out), and further exposure will lead to disorientation, amnesia, hearing loss, and eventually loss of consciousness.

EFEK GAYA PERCEPATAN LINIER PADA TUBUH

Gaya percepatan dalam penerbangan luar angkasa.

Percepatan sentrifugal (-), kec. Berputar-putar abnormal

Percepatan saat peluncuran dan perlambatan pada saat pendaratan dapat merupakan persoalan serius.

So what are the physiological effects of high G? During vertical acceleration, the body's thoracic and carotid pressure sensors signal the sympathetic nervous system to compensate for the lowering of blood pressure, by increasing heart rate, increasing heart stroke volume, and constricting muscles so as to reduce the volume of arteries. A lack of oxygen to the brain can cause cerebral hypoxia, but there's a lag time of around 5 seconds before brain function is impaired. The response of the sympathetic nervous system takes around 12 seconds, which is twice as long as the cerebral hypoxia reserve time, so brain damage is likely under high vertical G. This nerve reaction is automatically engaged by the 'fight or flight' response of the body's endocrine system, which becomes important in sustained high G environments. Vertical G also causes blood to pool at the bottom of the lungs where the alveolae are also shrunk or collapsed under differential pressure, oxygen absorption is decreased and lungs may collapse (Davis et al 2008)

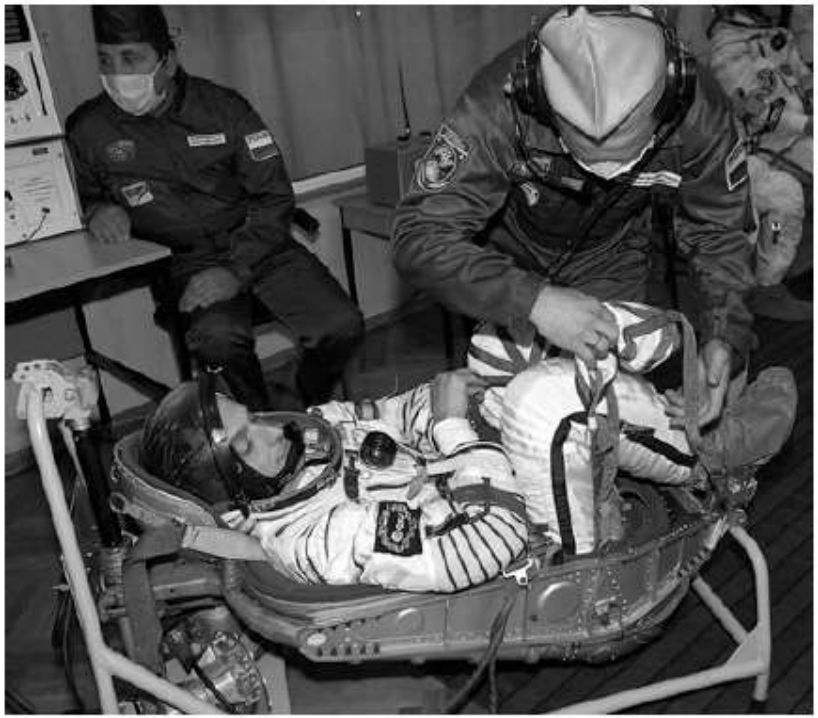
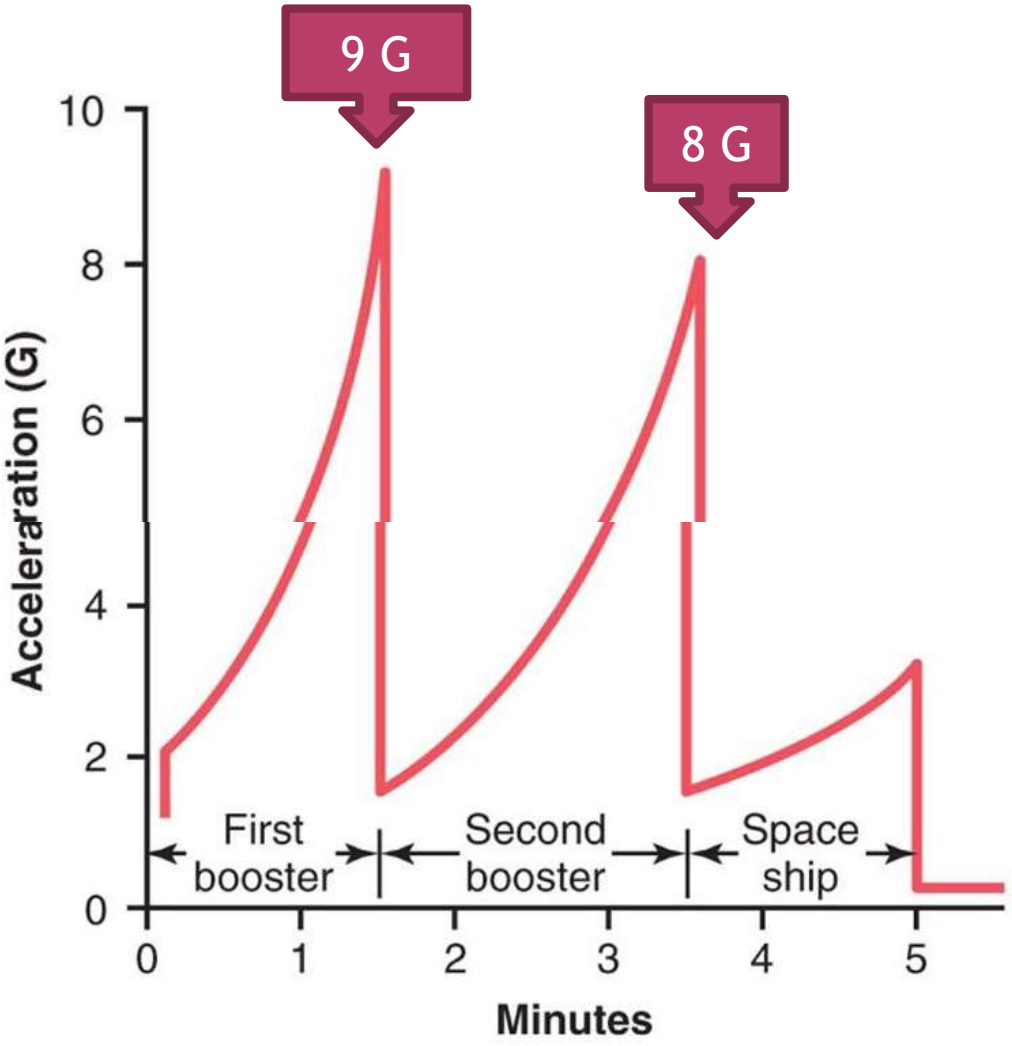


Fig. 2 The horizontal position assumed by astronauts for launch and re-entry (Credit: Clement 2011)

GAYA PERCEPATAN PADA WAKTU PESAWAT LUAR ANGKASA MELAKUKAN PELUNCURAN



- **Posisi berdiri**, tubuh tdk dpt bertahan terhadap percepatan linear sebesar ini.
- **Posisi setengah berbaring** tegak lurus terhadap sumbu percepatan, tubuh dpt bertahan

KEADAAN TANPA BOBOT DI RUANG ANGKASA

Di luar angkasa gravitasi
= G Nol = gravitasi mikro
(melayang di dalam ruangan)

Masalah pada keadaan tanpa bobot

Mabuk perjalanan (50%) space motion sickness

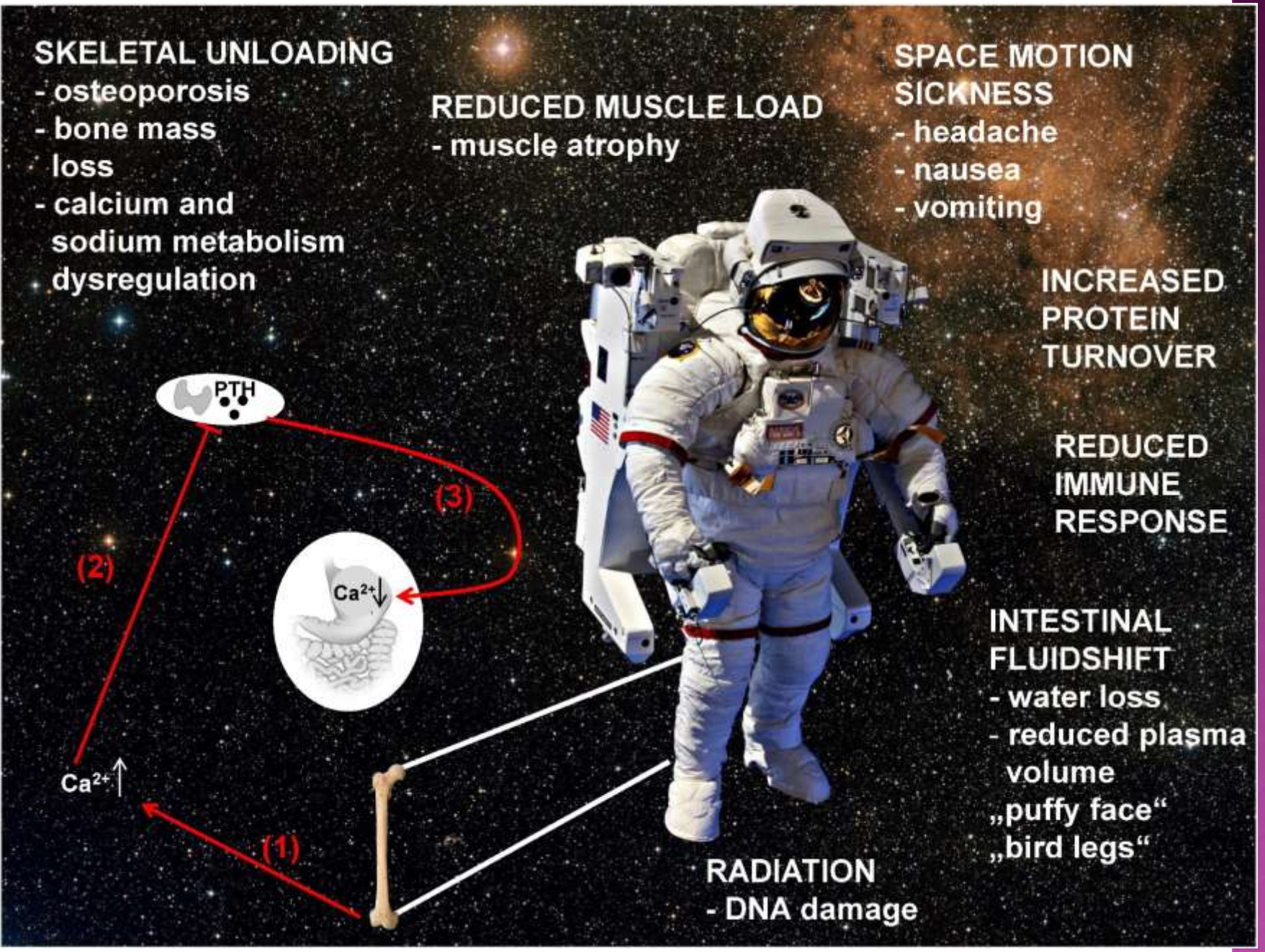
- Pola sinyal pergerakkan yg tdk umum di pusat keseimbangan otak dan tdk terdapat sinyal-sinyal gravitasional.

Translokasi cairan tubuh akibat kegagalan gravitasi menimbulkan tekanan hidrostatik

- Volume darah, massa sel eritrosit, dan curah jantung menurun

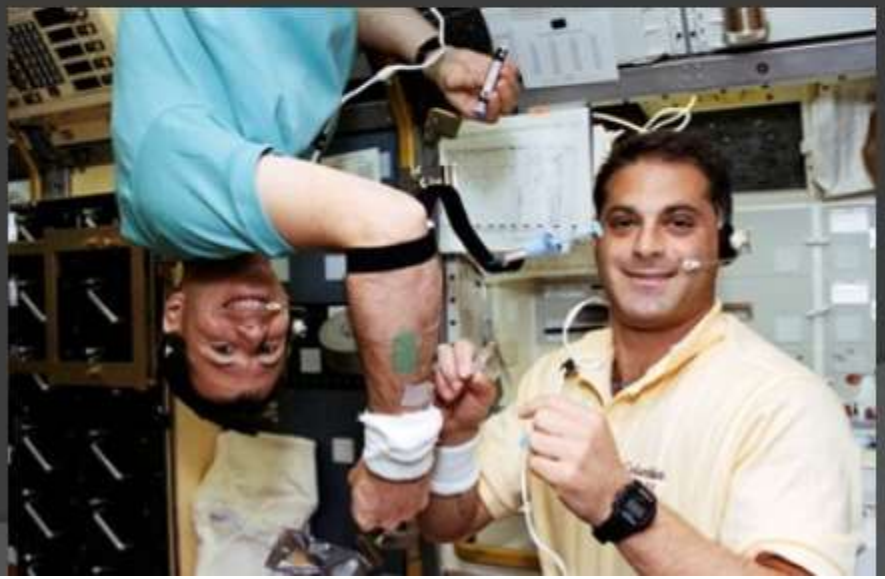
Aktifitas fisik (-) kekuatan otot tdk diperlukan u/melawan gravitasi

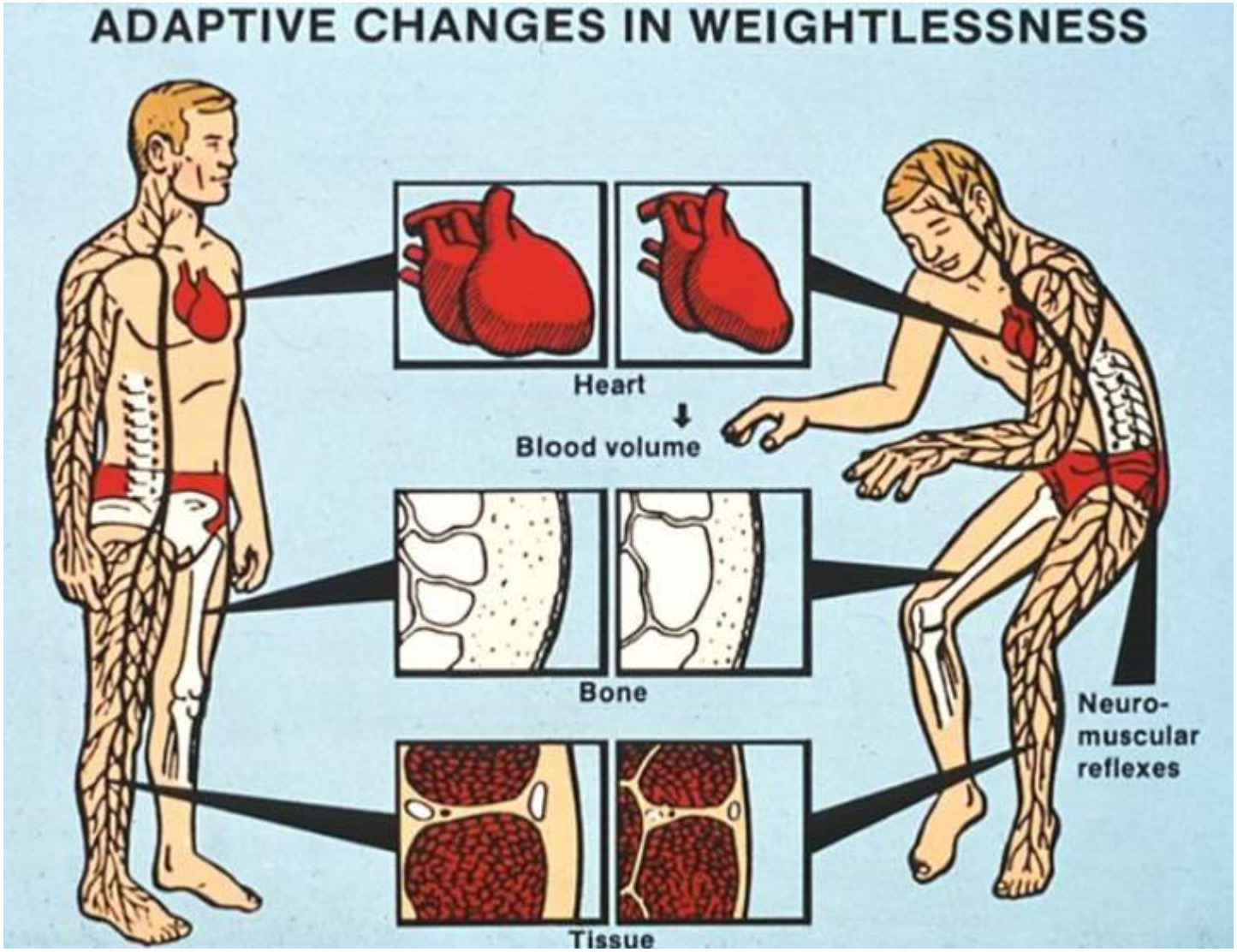
- Hilangnya Ca dan fosfat
- Kekuatan otot dan kapasitas kerja berkurang


SKELETAL UNLOADING
- osteoporosis
- bone mass loss
- calcium and sodium metabolism dysregulation
REDUCED MUSCLE LOAD
- muscle atrophy
SPACE MOTION SICKNESS
- headache
- nausea
- vomiting
INCREASED PROTEIN TURNOVER
REDUCED IMMUNE RESPONSE
INTESTINAL FLUIDSHIFT
- water loss
- reduced plasma volume
„puffy face“
„bird legs“
RADIATION
- DNA damage
PTH
(1)
(2)
(3)
Ca2+
Ca2+

Effects on the Body

- Bone Loss
- Decrease in muscle tissue
- Decrease in heart size
- Puffy-Head Bird Leg Syndrome




ADAPTIVE CHANGES IN WEIGHTLESSNESS
Heart
Blood volume
Bone
Tissue
Neuro-
muscular
reflexes

RADIATION

- Luar angkasa tidak memiliki atmosphere.
- Luar angkasa tidak memiliki atmospheric filter untuk melindungi manusia dari eksposure radiasi

↓

Kerusakan DNA

PENURUNAN KONDISI SISTEM KARDIOVASKULAR, OTOT, DAN TULANG SELAMA TERPAJAN KEADAAN TANPA BOBOT YANG LAMA

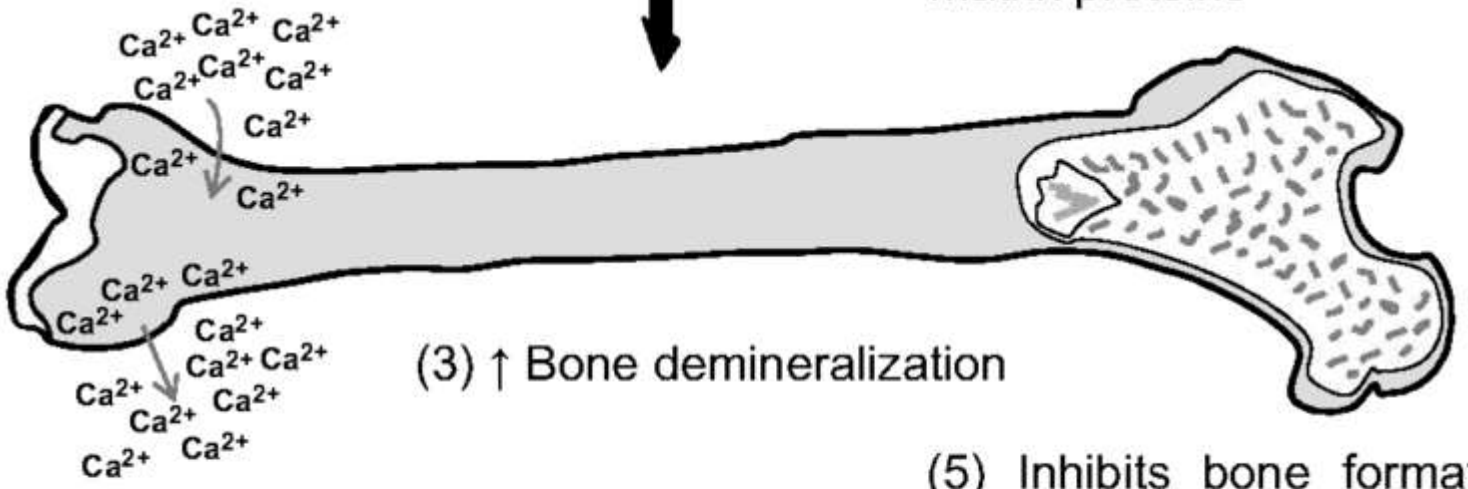
Bones

- Bone is living tissue.
- Bone is formed by cells called osteoblasts.
- Osteoclasts, large multinucleate cells, break down old bone and are responsibly for releasing calcium into the blood stream.
- On Earth the process is in equilibrium.

Microgravity

(1) ↓ Ca^{2+} absorption

(4) ↓ mRNA levels for bone matrix proteins



(2) ↓ Ca^{2+} excretion in feces

(5) Inhibits bone formation and induces bone loss at cancellous sites

(6) Most dramatic bone loss in pelvis, lumber vertebrae, femoral neck

In Dr. Almeida's latest study, 16-week-old female C57BL/6J mice were subjected to 15-days of microgravity during the space shuttle Discovery's STS-131 mission in April, 2010. During this mission, the NASA team investigated changes in molecular gene expression and cellular mechanisms in the mice during their two weeks in orbit.

Following the animals' return, the investigators discovered the following changes when compared with a synchronous control group that were housed in identical Animal Enclosure Module (AEM) habitats but remained on Earth:

1. Decreased bone volume

2. Decreased bone thickness

3. 197% increase in the number of osteoclasts (a marker for osteoclastic bone degeneration)

4. Lacunar osteolysis

5. Upreguation of matrix metalloproteinases (MPP) (a marker for osteocytic osteolysis)

6.Cell cycle inhibition of osteoblasts

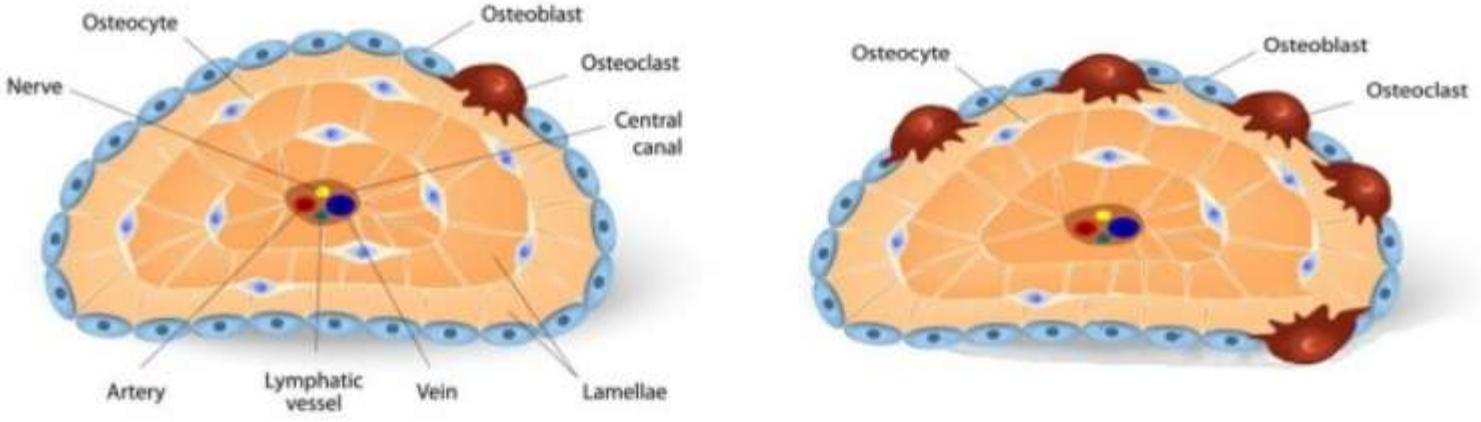


Figure 1. Cross-sectional diagram of a femur demonstrating the location and morphology of oseteoclasts, osteoblasts and osteocytes that participate in bone remodeling on Earth (left) and in space (right).

These studies show that, as observed in astronauts, the pelvic and femoral bones in mice exposed to weightlessness rapidly lose mass and that both osteocyte and osteoblast numbers are reduced. In sum, microgravity-associated bone loss in C57BL/6J mice is not limited to osteoclastic degradation, but also involves osteoblastic and osteocytic degenerative responses.

Figure 2. The effects of bone loss are illustrated on the left panel. Astronaut Wakata displays a bisphosphonate distribution card. He and his colleagues take bisphosphonate once a week and exercise to prevent bone loss and decrease the risk of bone fractures while aboard the International Space Station. (Images courtesy of NASA).

BERKURANGNYA MASSA OTOT: AKIBAT BURUK PENERBANGAN LUAR ANGKASA

Astronot wanita (3) dan astronot pria (4)

↓

Penurunan drastis dan signifikan massa otot-otot penyangga beban tubuh

↓

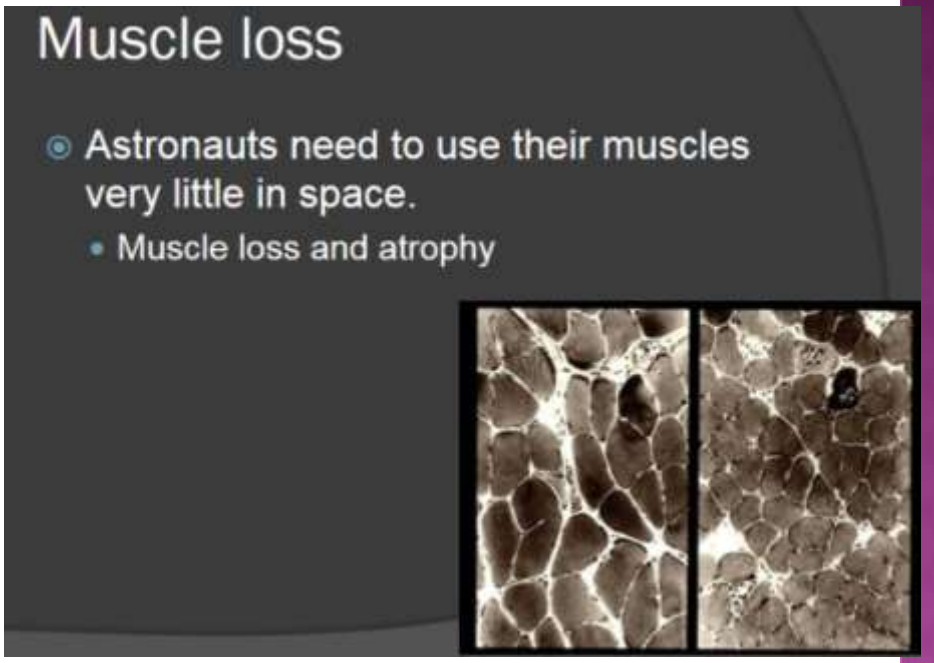
Upaya untuk menggerakan tubuh di luar angkasa lbh ringan drpd bumi

↓

Tidak dibutuhkan tegangan otot aktif yg melawan gravitasi

↓

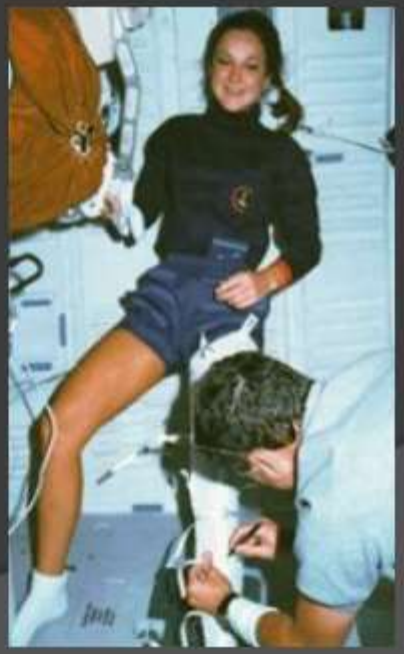
Disebut atrofi fungsional



← Otot-otot yg paling terkena adalah otot yg menopang tubuh melawan gravitasi.

Fluid Shift

- Body no longer experiences the downward pull of gravity to distribute blood and other body fluids.
- Headward shift.



Super ficial tissue thickness in the **tibia decreases by 15%,** whereas it **increases by 7% in the forehead.**

Puffy face

Upper part: crew of Odissea Mission before launch,

lower part: crew in-flight, first day (Credit: European Space Agency).

Sleep