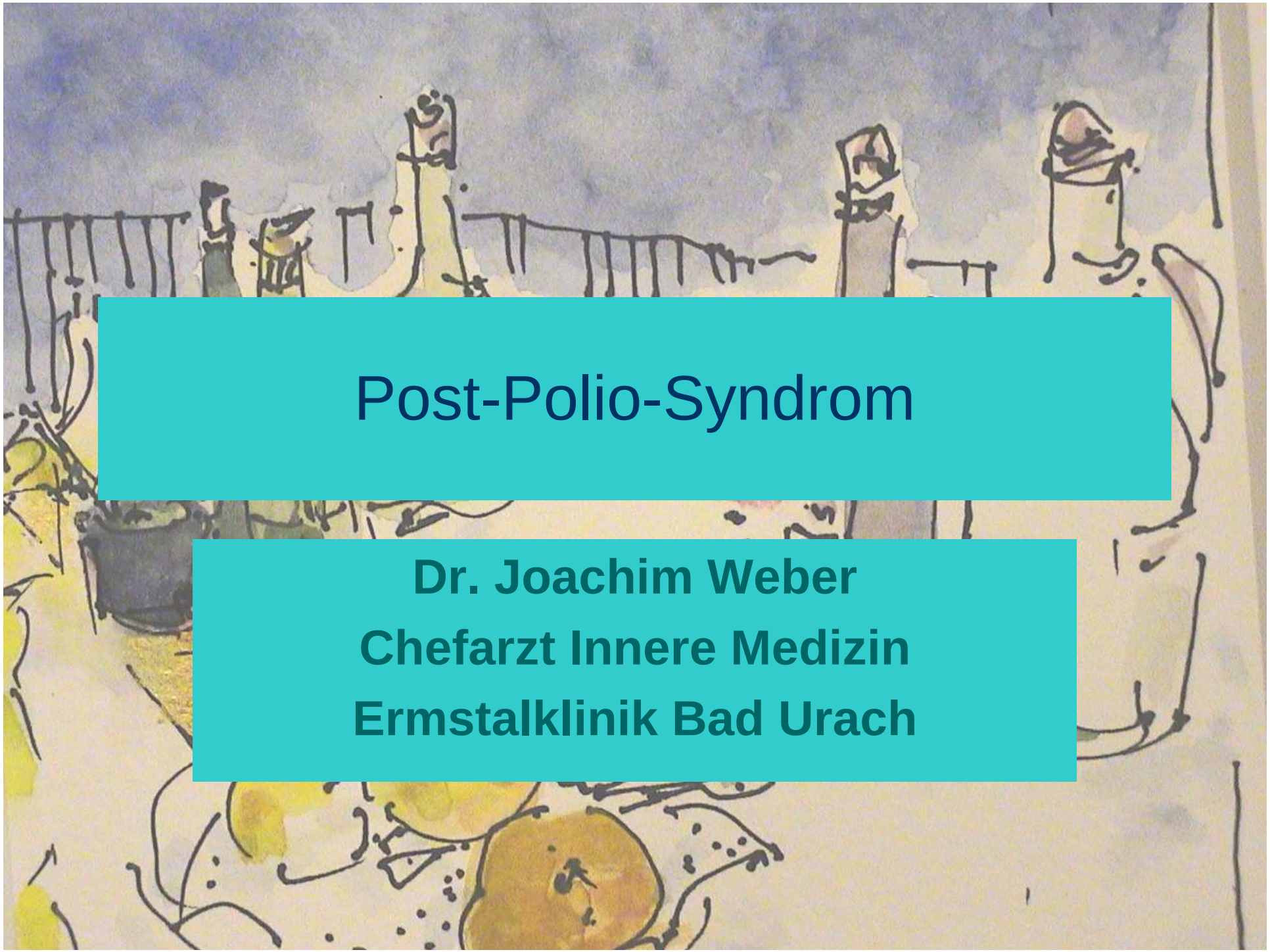
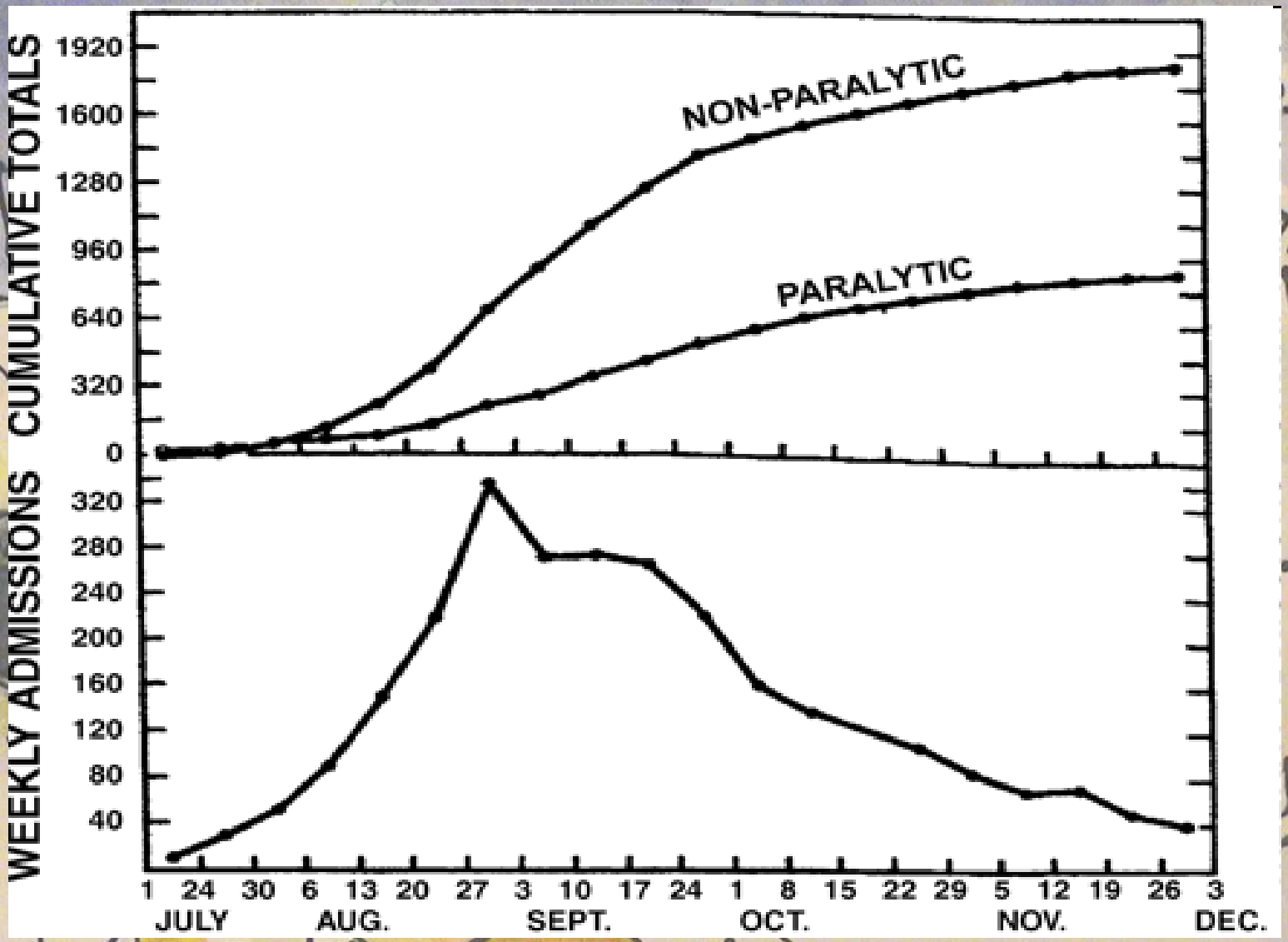


Post-Polio-Syndrom

Dr. Joachim Weber
Chefarzt Innere Medizin
Ermstalklinik Bad Urach


WEEKLY ADMISSIONS CUMULATIVE TOTALS
1920
1600
1280
960
640
320
0
NON-PARALYTIC
PARALYTIC
320
280
240
200
160
120
80
40
1 24 30 6 13 20 27 3 10 17 24 1 8 15 22 29 5 12 19 26 3
JULY AUG. SEPT. OCT. NOV. DEC.

5

1952: The Copenhagen Polio Epidemic and the Birth of Intensive Care

There are many reasons why people may end up on an intensive-care unit, most frequently following major operations, but also after head injury, septicaemia or respiratory failure from paralysis of the chest muscles. At any one time a patient can be hitched up to a dozen or more pieces of equipment: heart monitor, machines to measure the concentration of gases in the blood and the blood pressure, a pacemaker, a dialysis machine. It all looks, and is, so impressive that it can be difficult to appreciate that central to all this technological wizardry is just one piece of equipment, the ventilator blowing oxygen into the lungs. Oxygen alone ensures the heart carries on beating and 'buys time' for tissues to heal and the complications of impaired body function to be attended to. The indispensable role of oxygen in human physiology has been known for the best part of 200 years, but the appreciation of its central role in the survival of the critically ill starts abruptly with the Copenhagen polio epidemic of 1952.

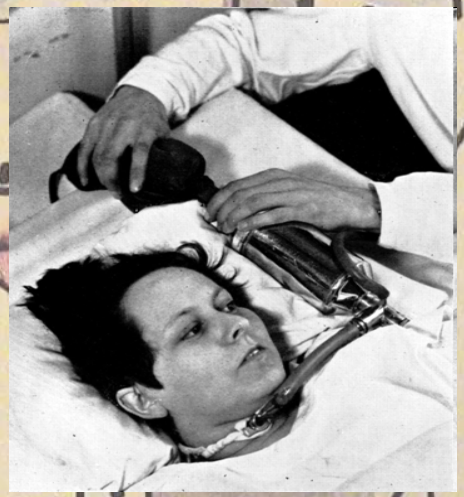
Eiserne Lunge

Geschichte und Entwicklung

Die Erfindung der Überdruckbeatmung

Björn Ibsen

Dänischer Anästhesist



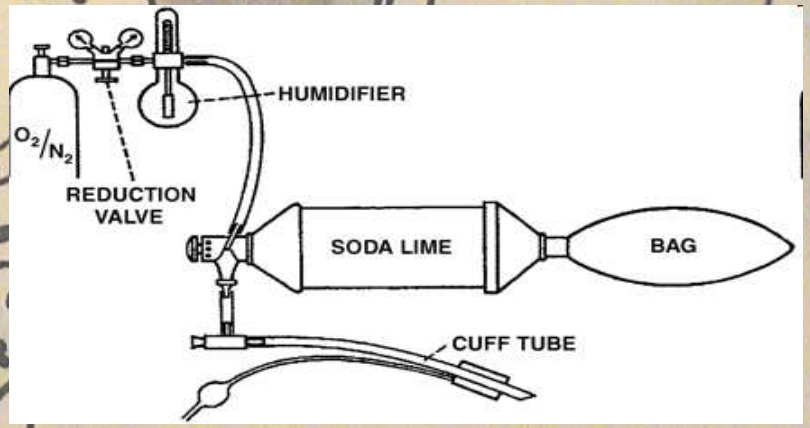


Table 1. *Blood pH and P_{co_2} values in a 5-yr-old boy after the onset of manual ventilation*

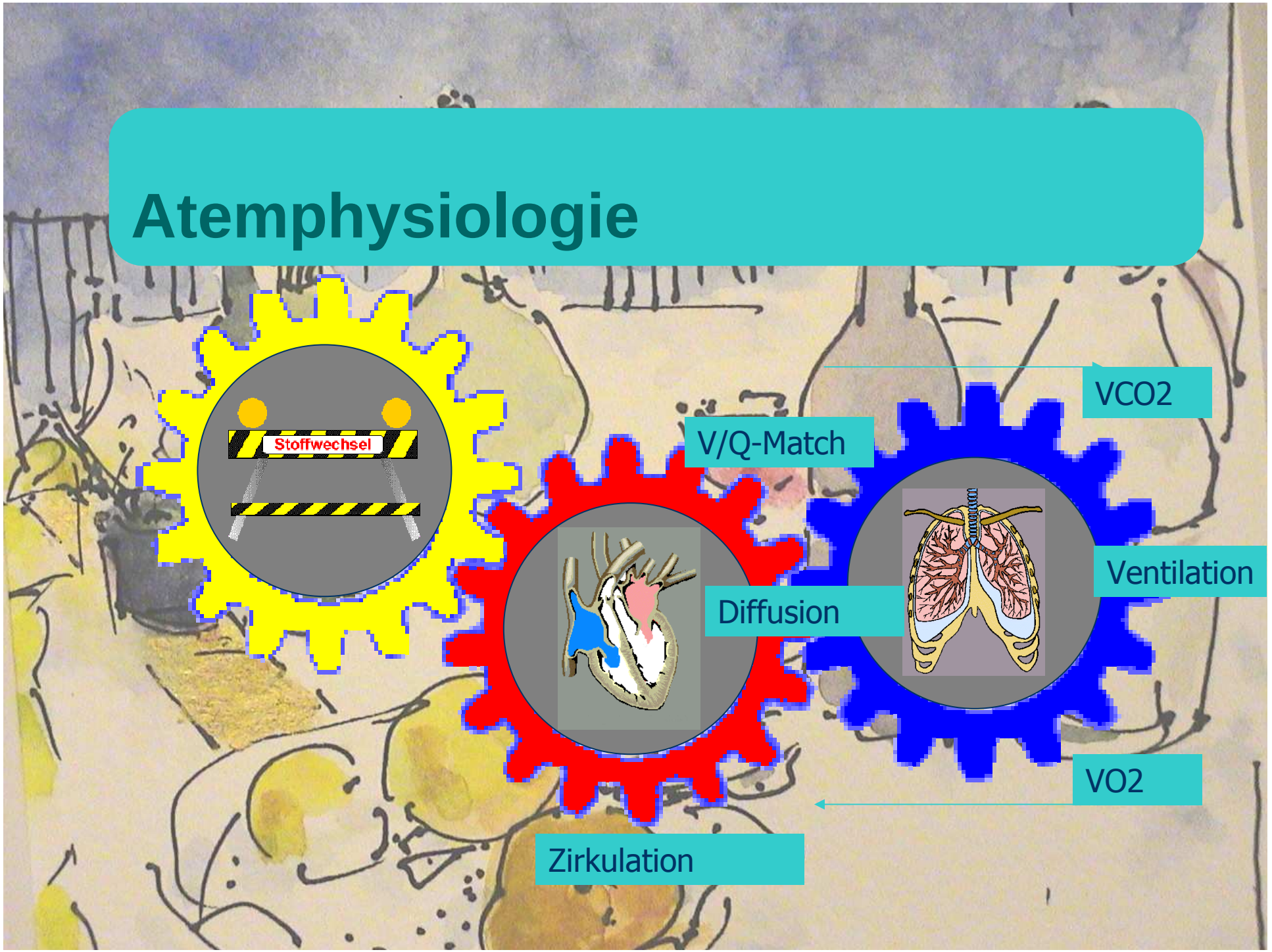
Hour	Blood	pH	PCO_2, mmHg	CO_2 conc, mmol	Bicarbonate, mmol
11:40 AM	venous	6.99	150	39.0	34.5
2:10 PM	arterial	7.52	32	24.4	24.5
3:05 PM	arterial	7.65	14	15.6	15.2

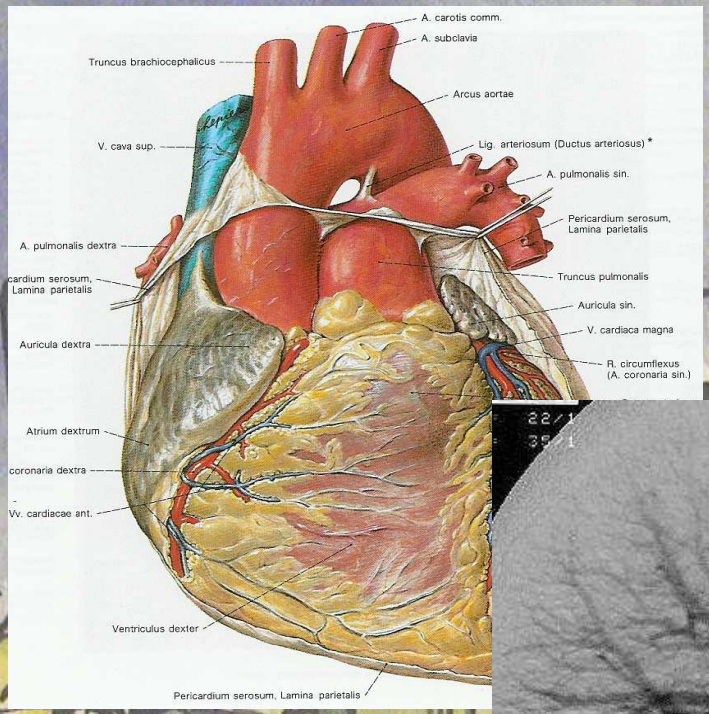
Blood pH and P_{CO_2} values in a 30-yr-old woman during manual ventilation over 13 days

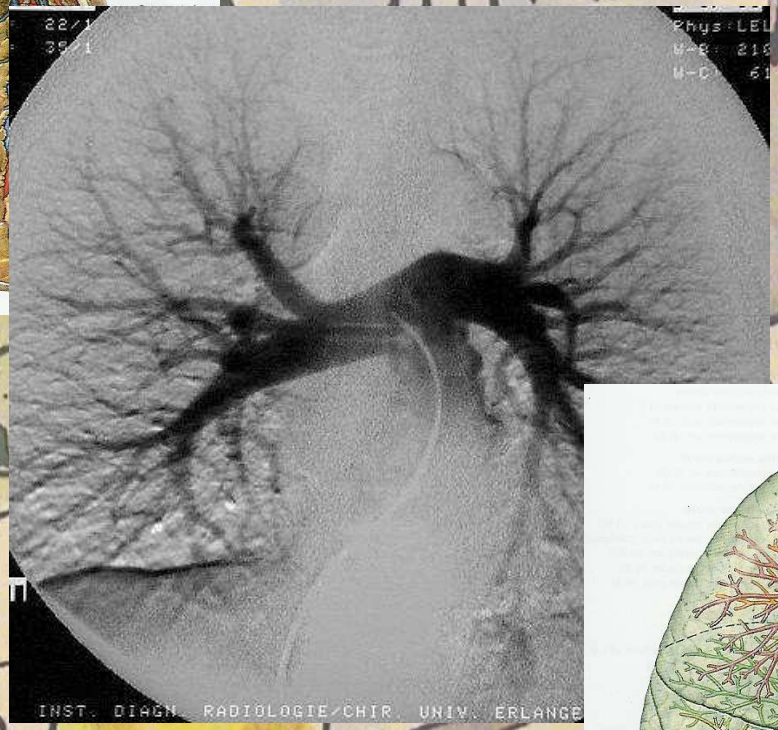
Date	Hour	Blood	pH	PCO_2, mmHg
Sept 1	1:00 PM	venous	7.49	32
	4:45 PM	"	7.47	31
2	9:15 AM	"	7.50	32
	10:30 AM	arterial	7.50	36
	10:35 AM	venous	7.47	34
	2:15 PM	"	7.48	36
4	9:10 AM	"	7.55	30
6	10:55 AM	arterial	7.55	30
8	12:35 PM	venous	7.56	31
10	10:10 AM	"	7.70	17
13	10:10 AM	"	7.56	23

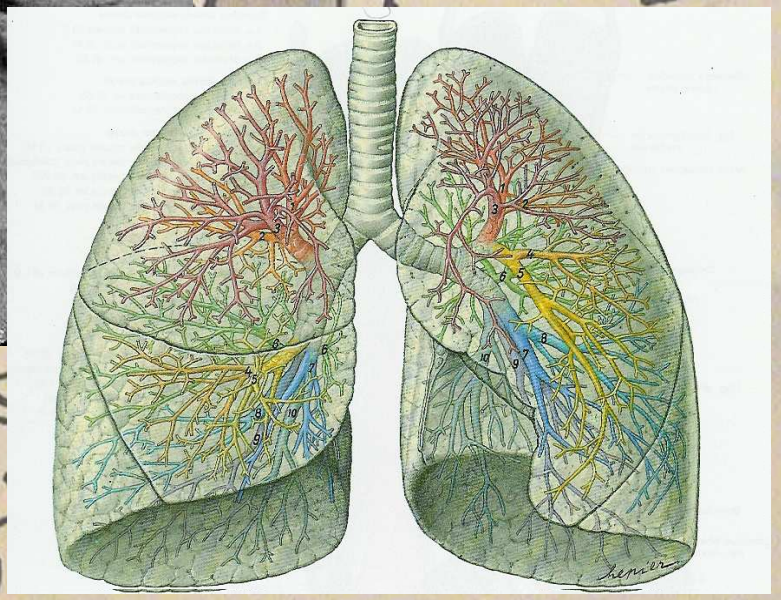
Geschichte und Entwicklung

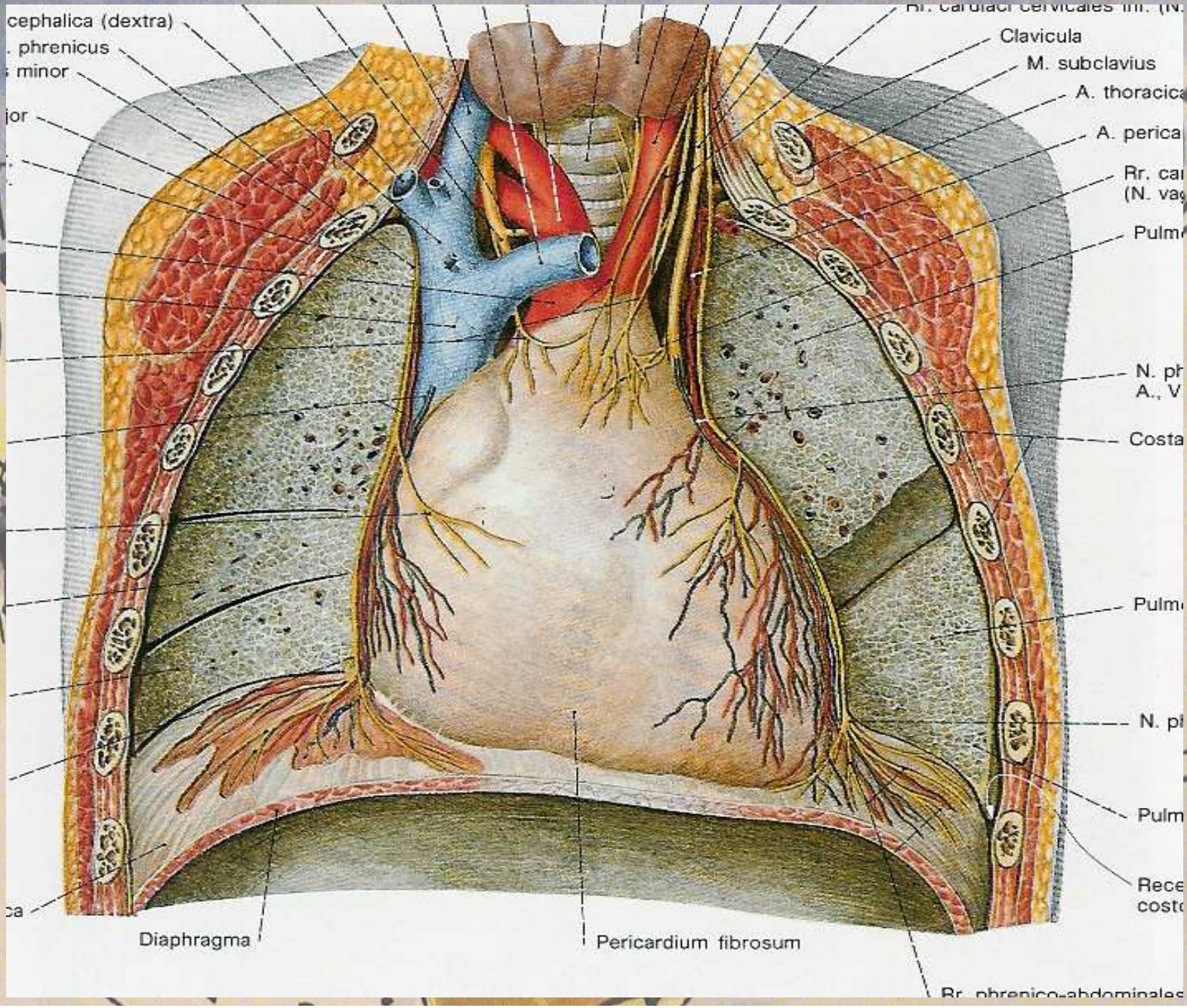
Atemphysiologie

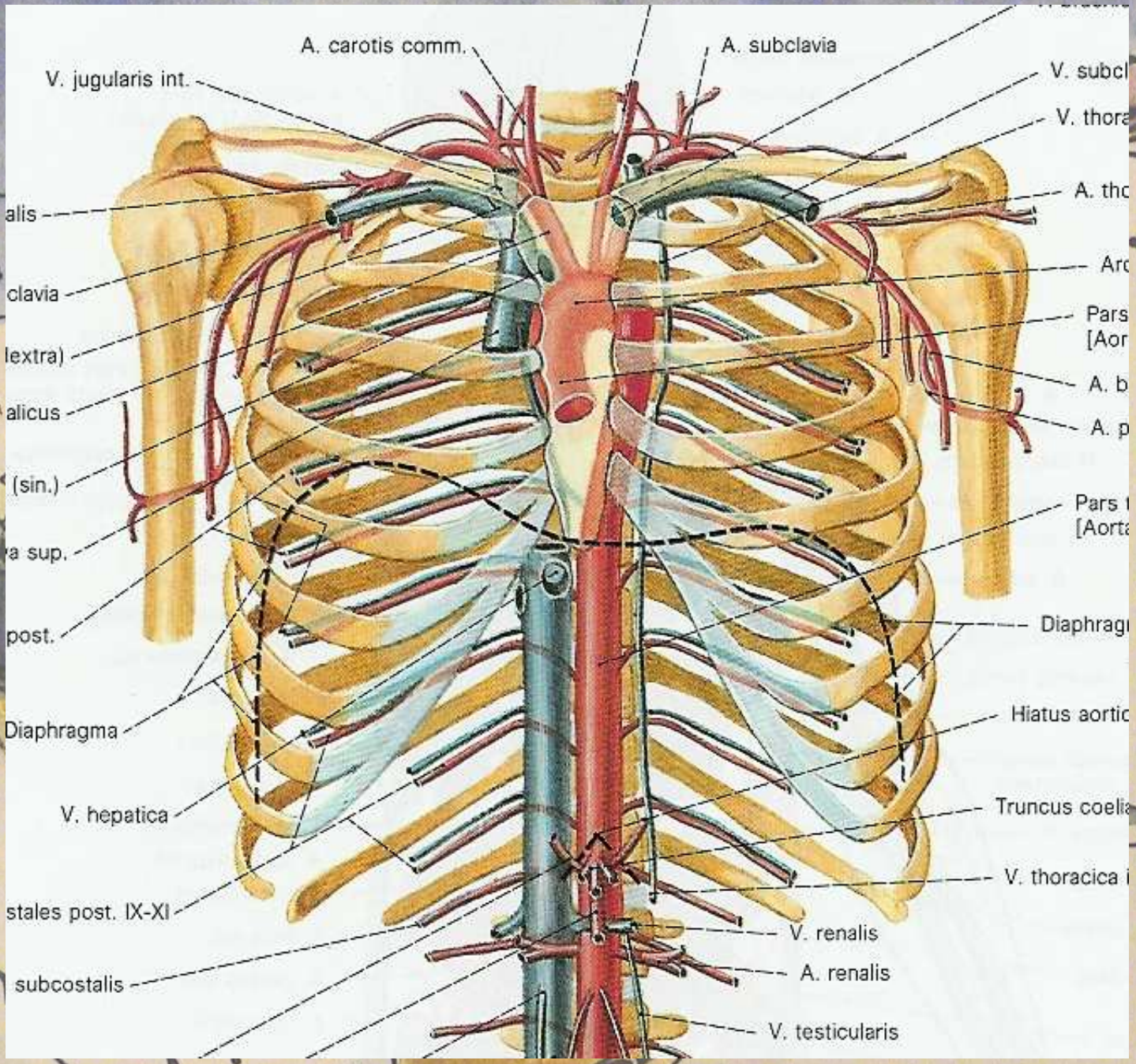



A. carotis comm.
A. subclavia
Truncus brachiocephalicus
Arcus aortae
V. cava sup.
Lig. arteriosum (Ductus arteriosus) *
A. pulmonalis sin.
Pericardium serosum, Lamina parietalis
A. pulmonalis dextra
cardium serosum, Lamina parietalis
Truncus pulmonalis
Auricula sin.
Auricula dextra
V. cardiaca magna
R. circumflexus (A. coronaria sin.)
Atrium dextrum
coronaria dextra
Vv. cardiacae ant.
Ventriculus dexter
Pericardium serosum, Lamina parietalis

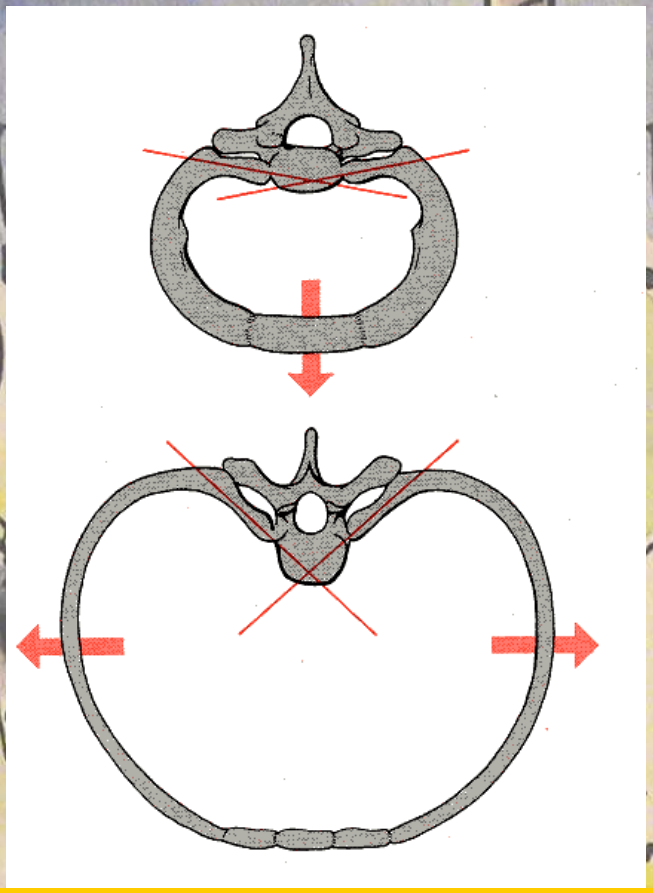

INST. DIAGN. RADIOLOGIE/CHIR. UNIV. ERLANGE

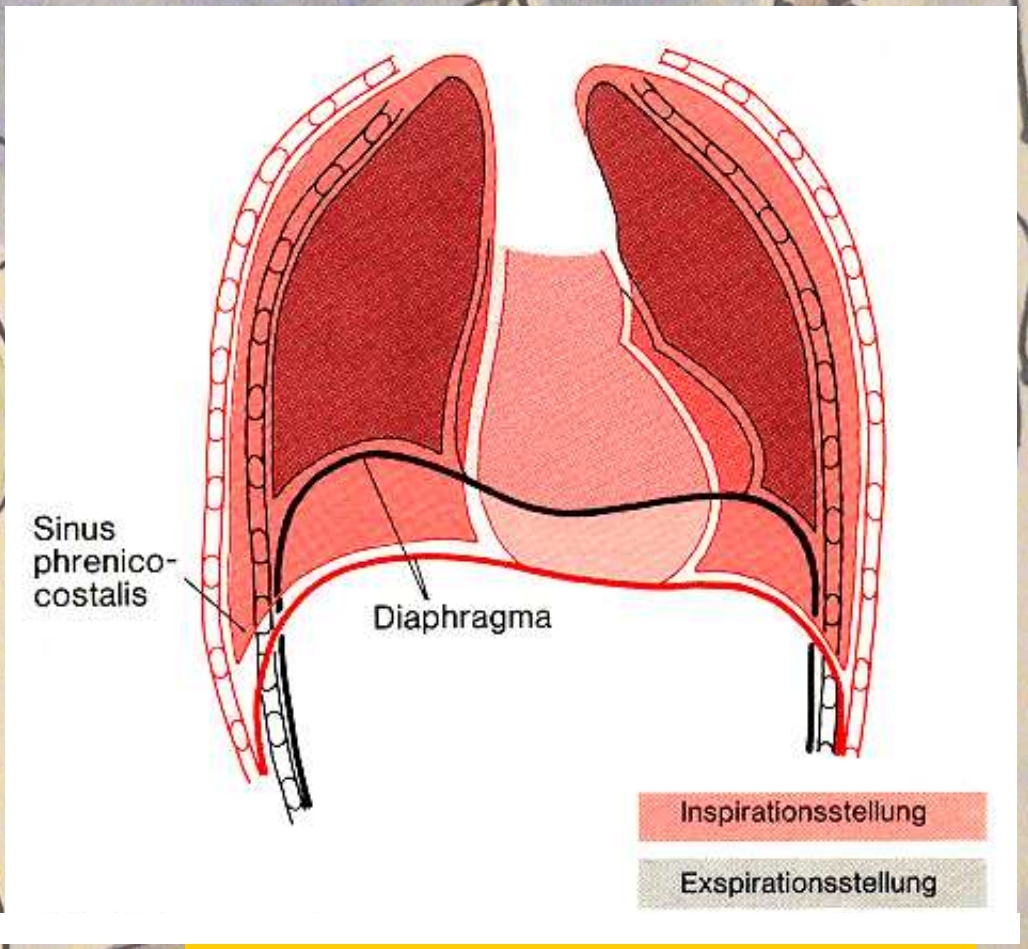



cephalica (dextra)
phrenicus
minor
Clavicula
M. subclavius
A. thoracica
A. perica
Rr. ca
(N. va
Pulm
N. ph
A., V
Costa
Pulm
N. p
Pulm
Rece
costo
Diaphragma
Pericardium fibrosum


A. carotis comm.
A. subclavia
V. jugularis int.
V. subcl
V. thora
A. tho
alis
clavia
lextra)
alicus
(sin.)
a sup.
post.
Diaphragma
V. hepatica
stales post. IX-XI
subcostalis
Arc
Pars
[Aor
A. b
A. p
Pars
[Aorta
Diaphragı
Hiatus aortic
Truncus coelia
V. thoracica i
V. renalis
A. renalis
V. testicularis

Thorax- und Zwerchfellatmung

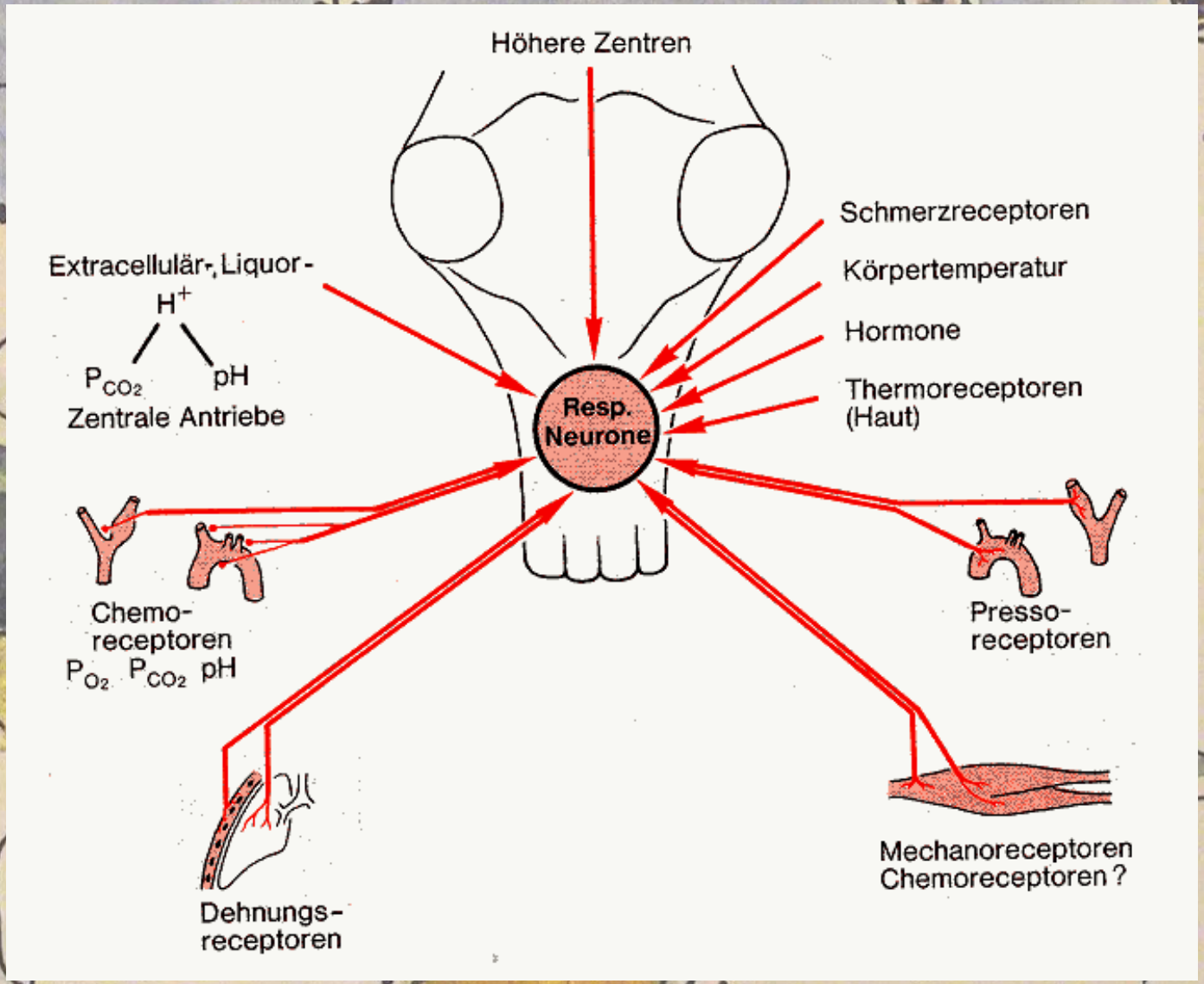




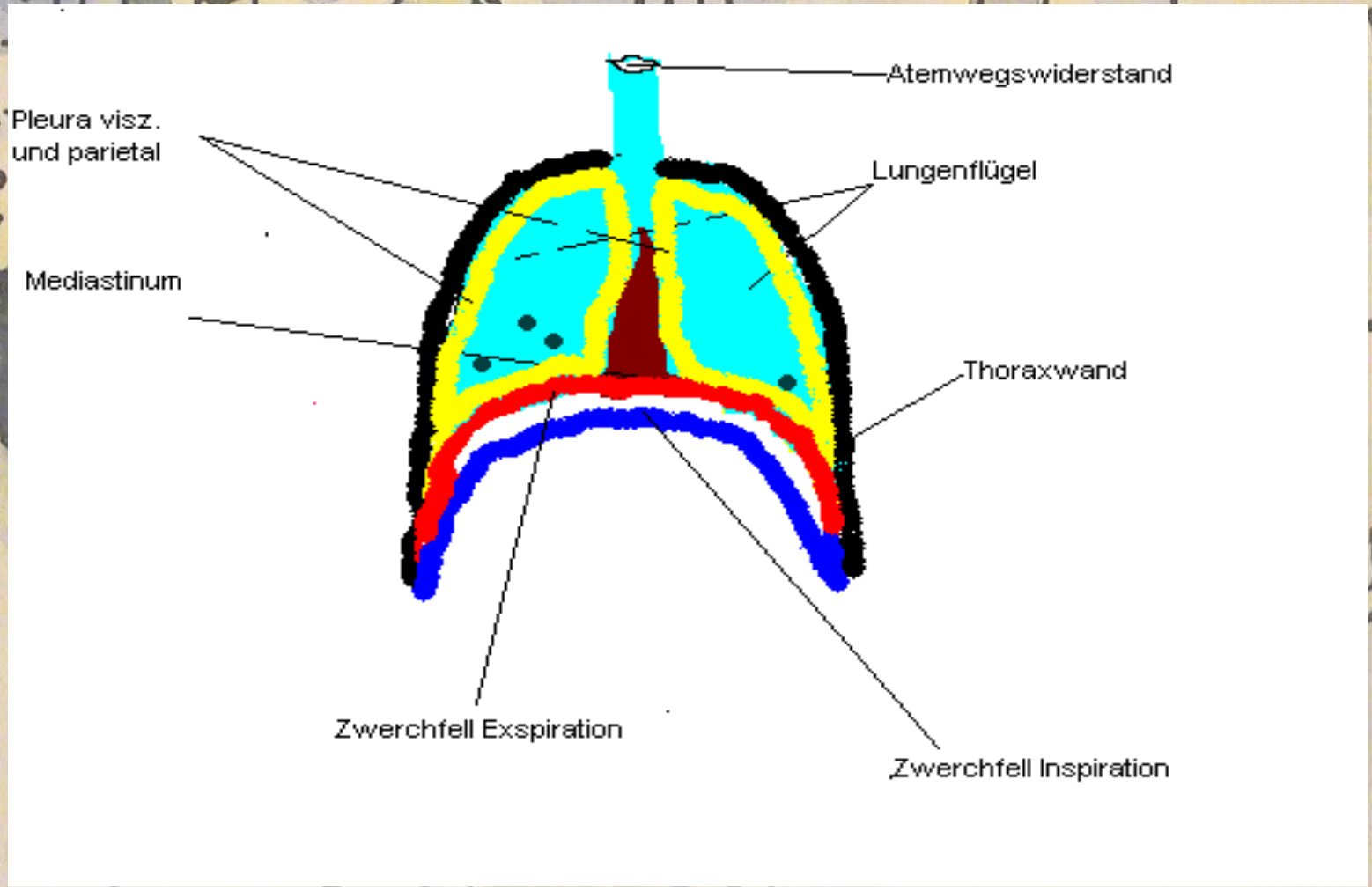
thorakale Atmung:
Zug nach cranial erweiter den Thorax → Inspiration. Zug nach caudal verengt den Thorax →Exspiration

abdominale Atmung: Abflachung des Zwerchfellserweitert den intrathorakalen Raum → Inspiration

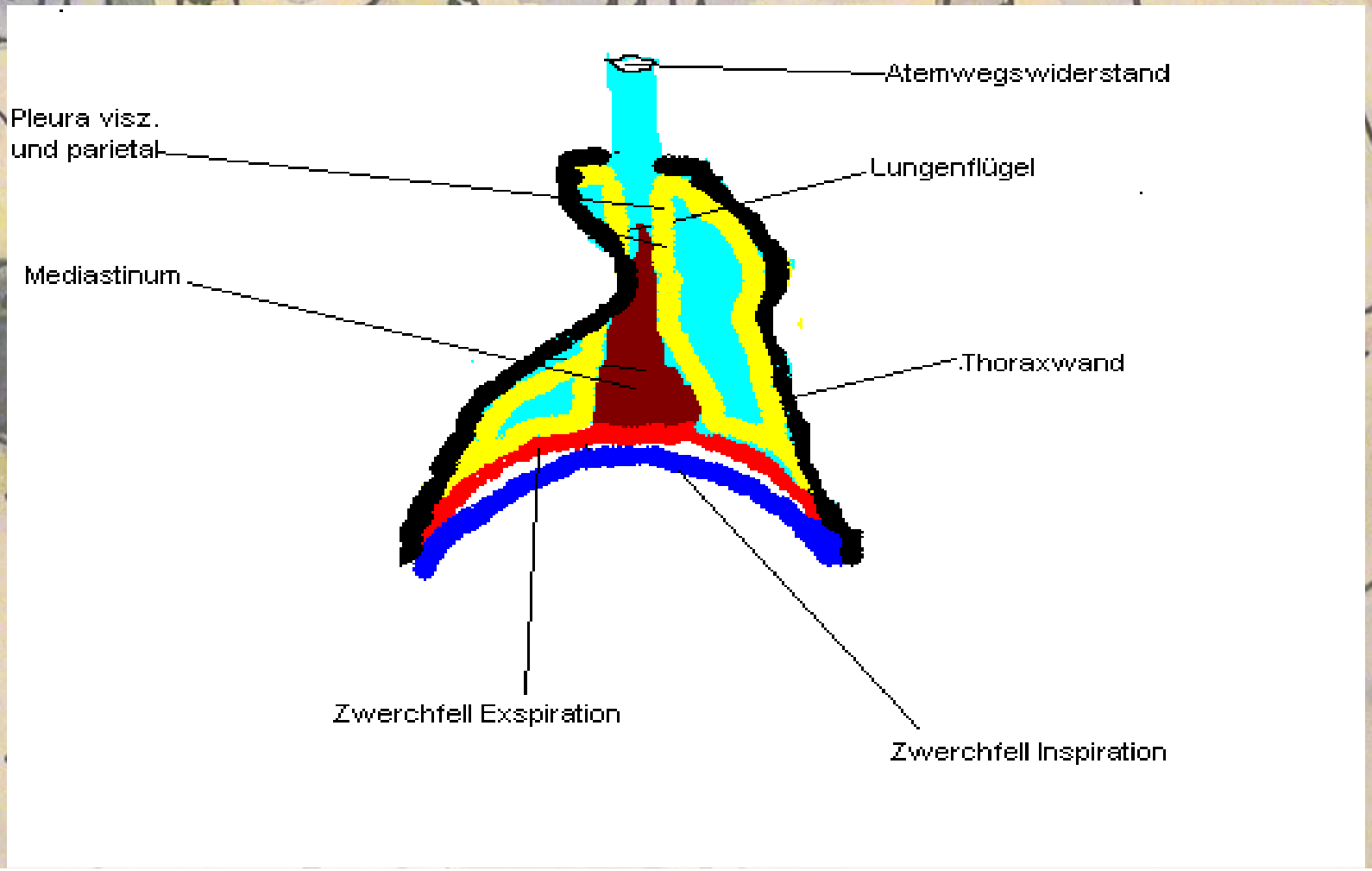
Einflüsse auf das Atemzentrum



Pathophysiologie der Ateminsuffizienz
Neurologische Erkrankungen



Pathophysiologie Skeletterkrankungen



PUBLICATION No. 51
Additional copies of this bulletin may be obtained without charge from The National Foundation for Infantile Paralysis, Inc.
120 Broadway
New York 5, N. Y.
BEekman 3-0500

Bulletin

SAVE THIS
You'll want it if infantile paralysis strikes.

When Polio Strikes . . . Helpful Hints for Everyone

June through September is the season when infantile paralysis generally is on the upswing in the United States. The National Foundation for Infantile Paralysis has compiled the following suggestions which will be helpful to residents of areas where poliomyelitis is on the march.

1. During an outbreak of infantile paralysis be alert to any early signs of illness or changes in normal state of health, especially in children. Do not assume that a stomach upset with vomiting, constipation, diarrhea, severe headache or signs of a cold and fever are of no importance. These may be among the first symptoms of infantile paralysis. All children and adults sick with unexplained fever should be put to bed and isolated pending medical diagnosis.

2. Don't delay calling a physician. Expert medical care given early may prevent many of the crippling deformities. Proper care from the onset may mean the difference between a life of crippling and normal recovery.

3. Today there is no known prevention or protection against infantile paralysis. All that can be done is to provide the best possible care. Your doctor, your health officer and your local Chapter of The National Foundation for Infantile Paralysis can and will do everything in their power to see to it that your community is ready to meet an epidemic.

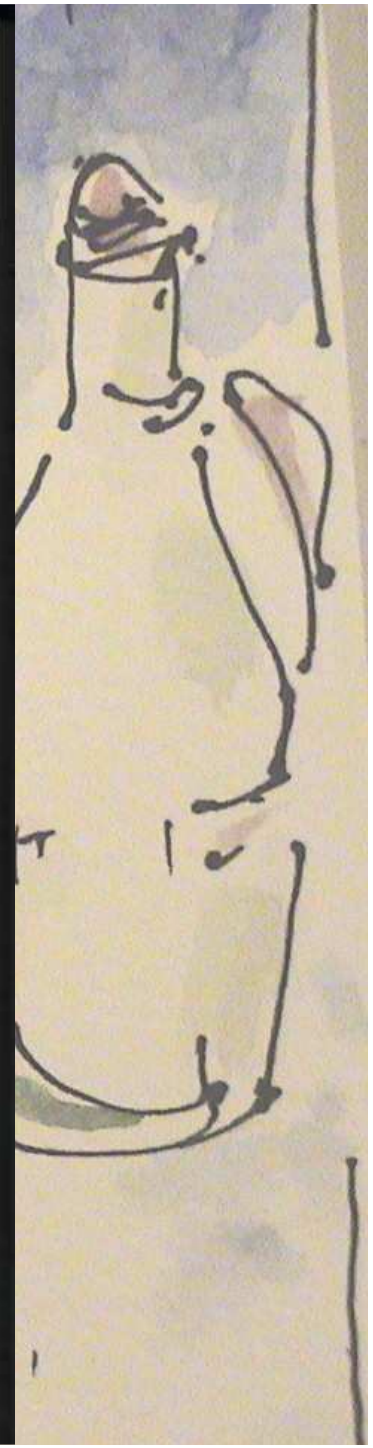
4. Observe these simple precautions:
   (a) Avoid overtiring and extreme fatigue from strenuous exercise.
   (b) Avoid sudden chilling such as would come from a plunge into extremely cold water on a very hot day.
   (c) Pay careful attention to personal cleanliness, such as thorough hand washing before eating. Hygienic habits should always be observed.
   (d) If possible avoid tonsil and adenoid operations during epidemics. Careful study has shown that such operations, when done during an epidemic, tend to increase the danger of contracting infantile paralysis in its most serious form.
   (e) Use the purest milk and water you can. Keep flies away from food. While the exact means of spread of the disease is not known, contaminated water and milk are always dangerous and flies have repeatedly been shown to carry the infantile paralysis virus.
   (f) Do not swim in polluted water.
   (g) Maintain community sanitation at a high level at all times.
   (h) Avoid all unnecessary contact with persons with any illness suspicious of infantile paralysis.

5. Don't become hysterical if cases do occur in your neighborhood. While infantile paralysis is communicable or catching during any outbreak, there are many who have such a slight infection that there are few or no symptoms. This large number of unrecognized infections is one of the reasons there is no practical way of preventing the spread of the disease. But it is also reassuring to know that, of the many persons who become infected, few develop serious illness and that, with good care, the majority who are stricken will make a satisfactory recovery. Remember that although this is a frightful disease, needless fear and panic only cause more trouble.

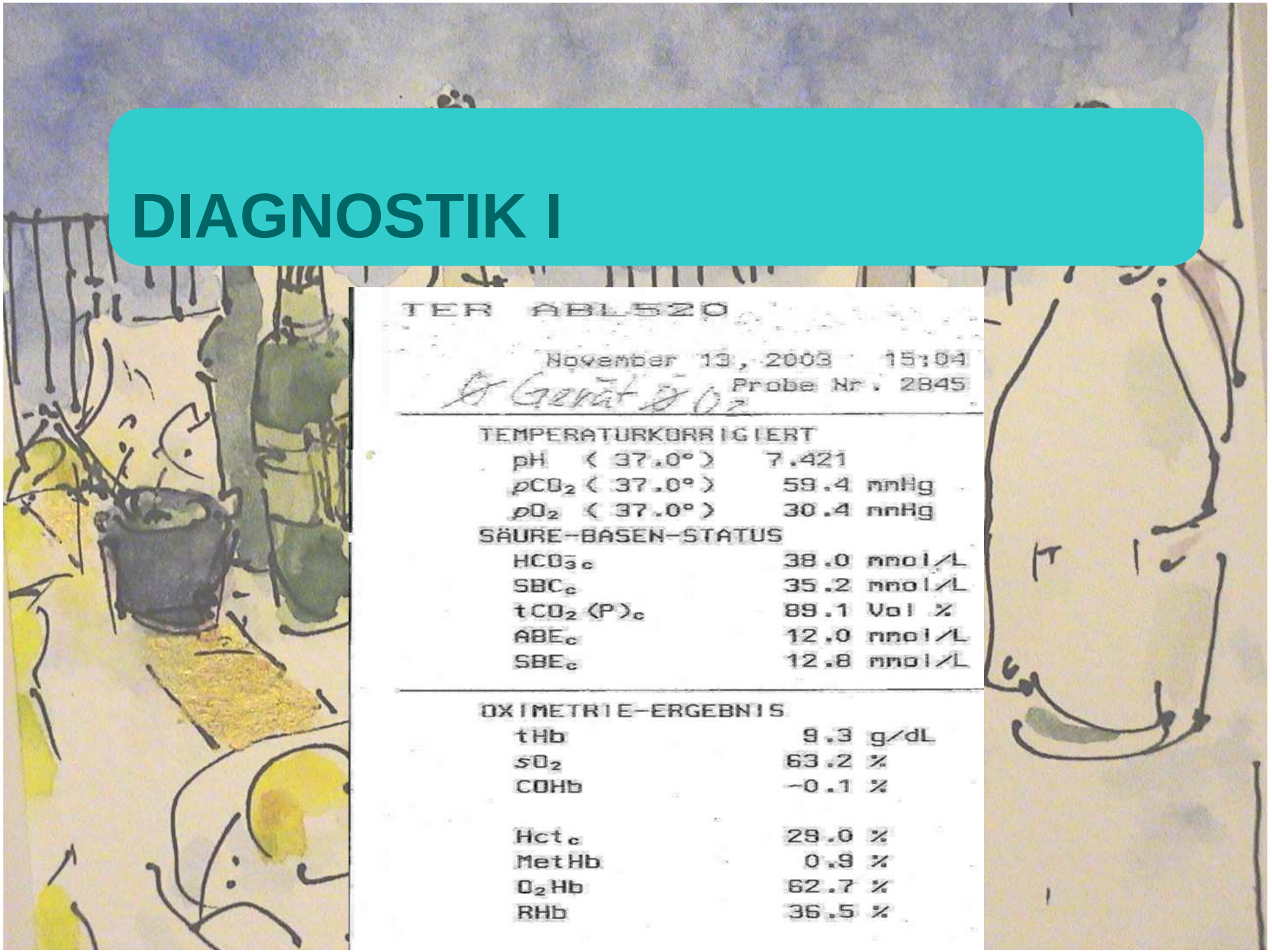
6. Attempts to stop the spread of the virus by closing places where people congregate have been uniformly unsuccessful. The resulting disturbance to community life is a disadvantage. Today there is no way by which the spread of infantile paralysis can be completely stopped.

7. There is no known cure for infantile paralysis. Good medical care will prevent or correct some deformities. But in about every fourth or fifth case there will be permanent paralysis that cannot be overcome. Do not believe those who for one reason or another promise to cure these cases. Be guided by sound medical advice if polio does strike in your family.

8. In almost all the counties of the United States there are local Chapters of The National Foundation for Infantile Paralysis prepared to help health officers, doctors, nurses, hospitals and patients in every way possible. These Chapters stand ready to assist the entire community. Know your Chapter — ask its help if needed — and volunteer to help your Chapter so that it will be able to render the necessary services.



DIAGNOSTIK I

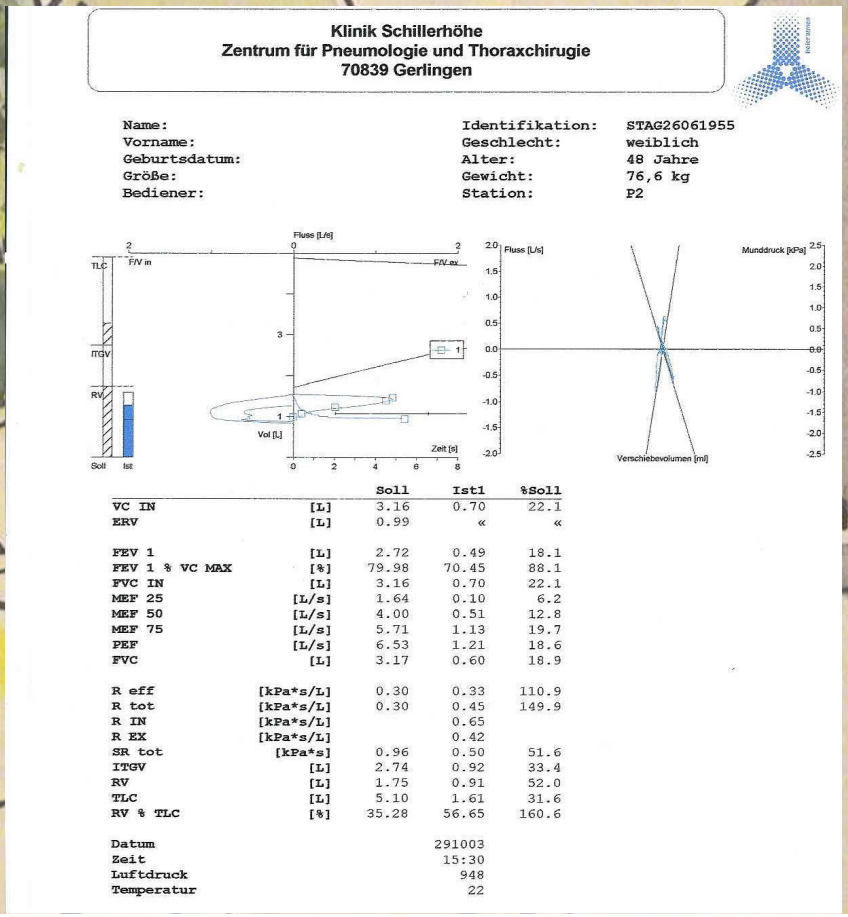


TER ABL520

November 13, 2003 15:04
Gerät Ø2 Probe Nr. 2845

TEMPERATURKORRIGIERT		
pH (37.0°)	7.421	
pCO_2 (37.0°)	59.4	mmHg
pO_2 (37.0°)	30.4	mmHg
SÄURE-BASEN-STATUS		
$HCO_3^-{}_c$	38.0	mmol/L
SBC_c	35.2	mmol/L
$tCO_2(P)_c$	89.1	Vol %
ABE_c	12.0	mmol/L
SBE_c	12.8	mmol/L
OXIMETRIE-ERGEBNIS		
tHb	9.3	g/dL
sO_2	63.2	%
COHb	-0.1	%
Hct_c	29.0	%
MetHb	0.9	%
O_2Hb	62.7	%
RHb	36.5	%

DIAGNOSTIK II

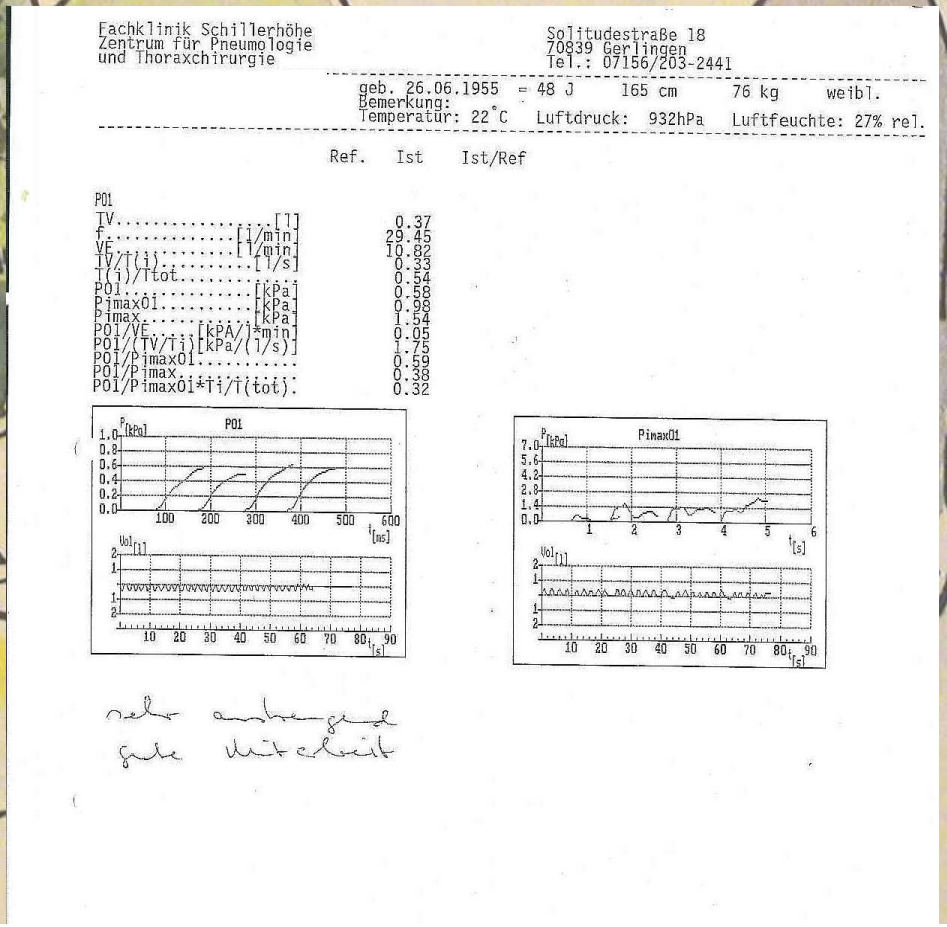


Klinik Schillerhöhe
Zentrum für Pneumologie und Thoraxchirurgie
70839 Gerlingen

Name:		Identifikation:	STAG26061955
Vorname:		Geschlecht:	weiblich
Geburtsdatum:		Alter:	48 Jahre
Größe:		Gewicht:	76,6 kg
Bediener:		Station:	P2

		Soll	Ist1	%Soll
VC IN	[L]	3.16	0.70	22.1
ERV	[L]	0.99	«	«
FEV 1	[L]	2.72	0.49	18.1
FEV 1 % VC MAX	[%]	79.98	70.45	88.1
FVC IN	[L]	3.16	0.70	22.1
MEF 25	[L/s]	1.64	0.10	6.2
MEF 50	[L/s]	4.00	0.51	12.8
MEF 75	[L/s]	5.71	1.13	19.7
PEF	[L/s]	6.53	1.21	18.6
FVC	[L]	3.17	0.60	18.9
R eff	[kPa*s/L]	0.30	0.33	110.9
R tot	[kPa*s/L]	0.30	0.45	149.9
R IN	[kPa*s/L]		0.65	
R EX	[kPa*s/L]		0.42	
SR tot	[kPa*s]	0.96	0.50	51.6
ITGV	[L]	2.74	0.92	33.4
RV	[L]	1.75	0.91	52.0
TLC	[L]	5.10	1.61	31.6
RV % TLC	[%]	35.28	56.65	160.6
Datum			291003	
Zeit			15:30	
Luftdruck			948	
Temperatur			22	

DIAGNOSTIK III



Fachklinik Schillerhöhe
Zentrum für Pneumologie
und Thoraxchirurgie

Solitudestraße 18
70839 Gerlingen
Tel.: 07156/203-2441

geb. 26.06.1955 = 48 J 165 cm 76 kg weibl.
Bemerkung:
Temperatur: 22°C Luftdruck: 932hPa Luftfeuchte: 27% rel.

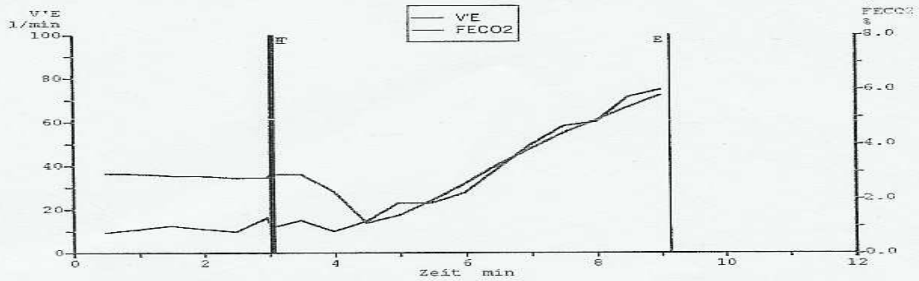
	Ref.	Ist	Ist/Ref
P01			
TV [l]		0.37	
f [l/min]		29.45	
VE [l/min]		10.82	
TV/Ti [l/s]		0.33	
Ti/Ttot		0.54	
P01 [kPa]		0.58	
PimaxÖI [kPa]		0.98	
Pimax [kPa]		1.54	
P01/VE [kPA/l*min]		0.05	
P01/(TV/Ti) [kPa/(l/s)]		1.75	
P01/PimaxÖI		0.59	
P01/Pimax		0.38	
P01/PimaxÖI*Ti/T(tot)		0.32	

sehr angespannte Mitarbeit

Klinik Schillerhöhe
Zentrum für Pneumologie und Thoraxchirurgie
70839 Gerlingen

Atemantrieb

Identifikation:		Station:	Schlaflabor
Name:		Größe:	181,0 cm
Vorname:		Gewicht:	101,0 kg
Geburtsdatum:		Alter:	73 Jahre
Geschlecht:		Bediener:	Li



Zeit min	VTex l	VTin l	FECO2 %	V'CO2 ml/min	BF 1/min	V'E 1/min	PaCO2 mmHg

Blutgasdaten-Eingabe Ruhe-BGA
Blutgase

Zeit	V'O2	PaCO2	PaO2	RER	CaCO2	CaO2	BE	AaDO2
00:25	275	35.50	66.30	0.76	–	–	–	–
00:30	0.523	0.694	2.94	213	18	9.3	35.50	
01:00	0.675	0.909	2.92	245	16	10.9	35.50	
01:30	0.730	0.900	2.85	278	17	12.6	–	
02:00	0.737	0.993	2.84	243	15	11.1	–	
02:30	0.592	0.794	2.77	210	17	9.8	–	
02:58	0.801	0.948	2.79	351	20	16.3	–	
Test								
03:30	0.870	1.024	2.91	338	17	15.0	–	
04:00	0.662	0.805	2.26	170	15	10.0	–	
04:30	0.835	0.984	1.12	123	18	14.7	–	
05:00	1.339	1.474	1.39	247	17	23.1	–	
05:30	1.229	1.339	1.93	340	19	23.0	–	
06:00	1.443	1.573	2.57	556	19	27.9	–	
06:30	1.909	2.104	3.25	973	20	38.7	–	
07:00	2.445	2.624	3.84	1471	20	49.6	–	
07:30	2.812	3.041	4.40	1981	21	58.0	54.80	
08:00	2.845	3.086	4.87	2271	21	60.2	54.80	

n = 6 – Ruhe – AMV = 11,7

Blutgasdaten-Eingabe TEst-Bga
Blutgase

Zeit	V'O2	PaCO2	PaO2	RER	CaCO2	CaO2	BE	AaD
08:17	-6648	54.80	130.40	-0.44	–	–	–	–
08:30	3.166	3.421	5.35	2967	23	71.5	54.80	
09:00	3.336	3.613	5.79	3363	22	74.9	54.80	

Ruhe-BGA
August 18, 2003 14:52
Probe Nr. 7226

TEMPERATURKORRIGIERT

pH (37.0°)	7.443	
pCO_2 (37.0°)	35.5	mmHg
pO_2 (37.0°)	66.3	mmHg

SÄURE-BASEN-STATUS

HCO_3^-c	23.9	mmol/L
SBC_c	25.0	mmol/L
$tCO_2(P)_c$	56.0	Vol %
ABE_c	0.8	mmol/L
SBE_c	0.3	mmol/L

OXIMETRIE-ERGEBNIS

tHb	15.6	g/dL
sO_2	93.0	%
COHb	0.5	%
Hct_c	47.8	%
MetHb	0.5	%
O_2Hb	92.1	%
RHb	6.9	%

August 18, 2003 15:27
Test-BGA Probe Nr. 7233

TEMPERATURKORRIGIERT

pH (37.0°)	7.303	
pCO_2 (37.0°)	54.8	mmHg
pO_2 (37.0°)	130.4	mmHg

SÄURE-BASEN-STATUS

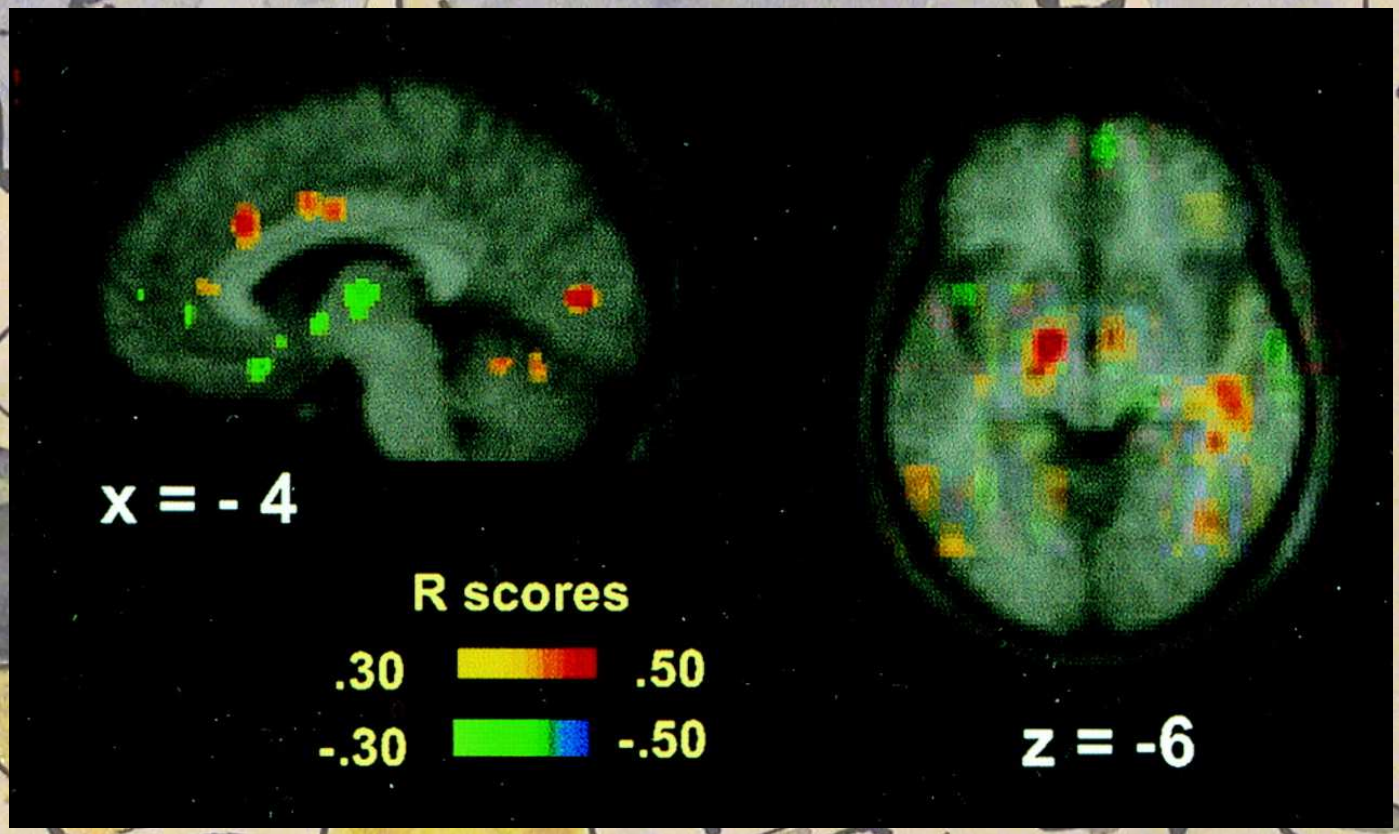
HCO_3^-c	26.3	mmol/L
SBC_c	23.7	mmol/L
$tCO_2(P)_c$	62.7	Vol %
ABE_c	-0.8	mmol/L
SBE_c	0.6	mmol/L

OXIMETRIE-ERGEBNIS

tHb	15.9	g/dL
sO_2	97.0	%
COHb	0.6	%
Hct_c	48.6	%
MetHb	0.4	%
O_2Hb	96.0	%
RHb	3.0	%

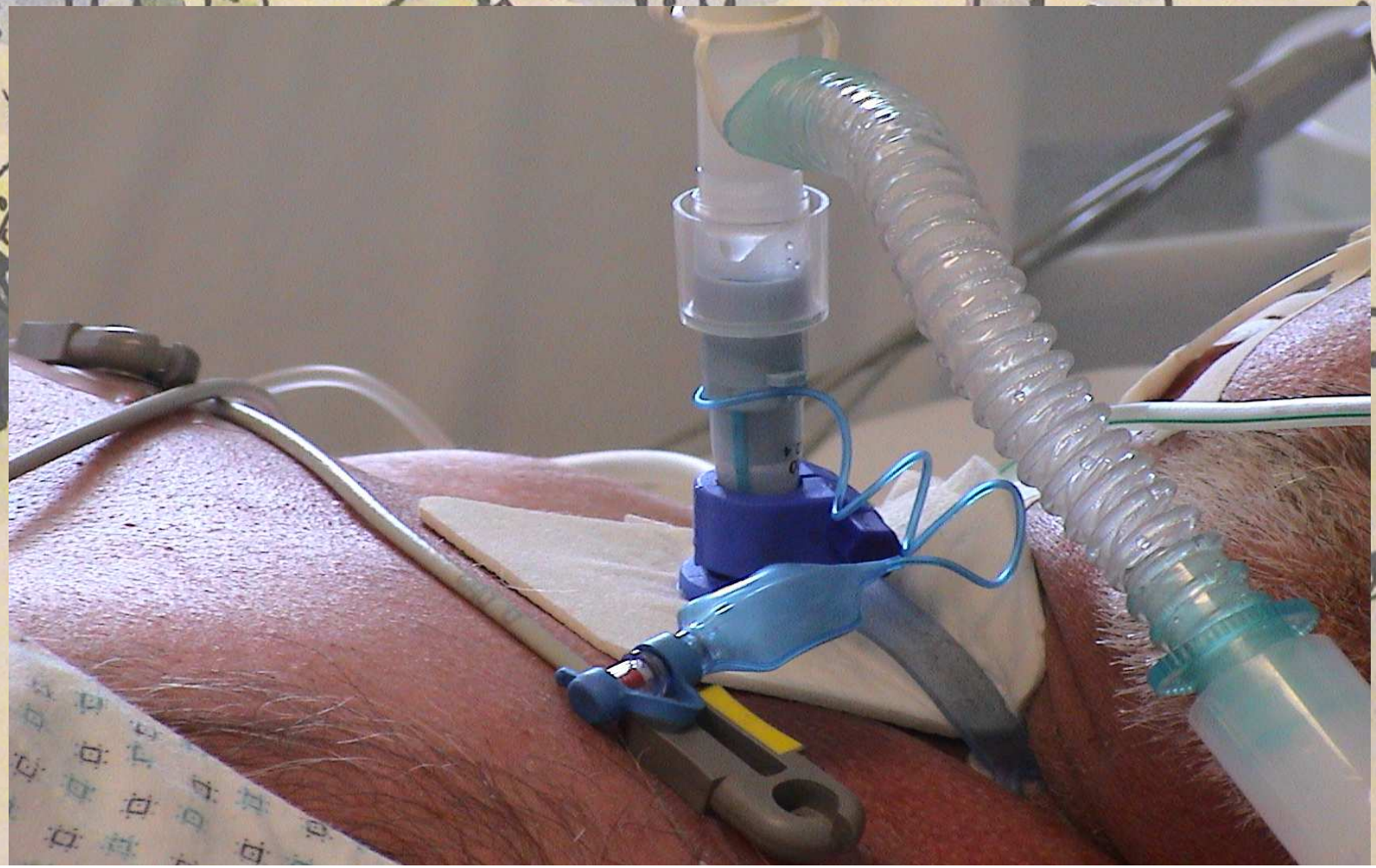
$$\frac{\Delta AMV\ (60{,}2 - 11{,}7)}{\Delta PCO_2\ (54{,}8 - 35{,}5)} = \frac{48{,}5 \times 1000}{19{,}3} = \underline{2513}\ ml/mmHg\ PCO_2$$

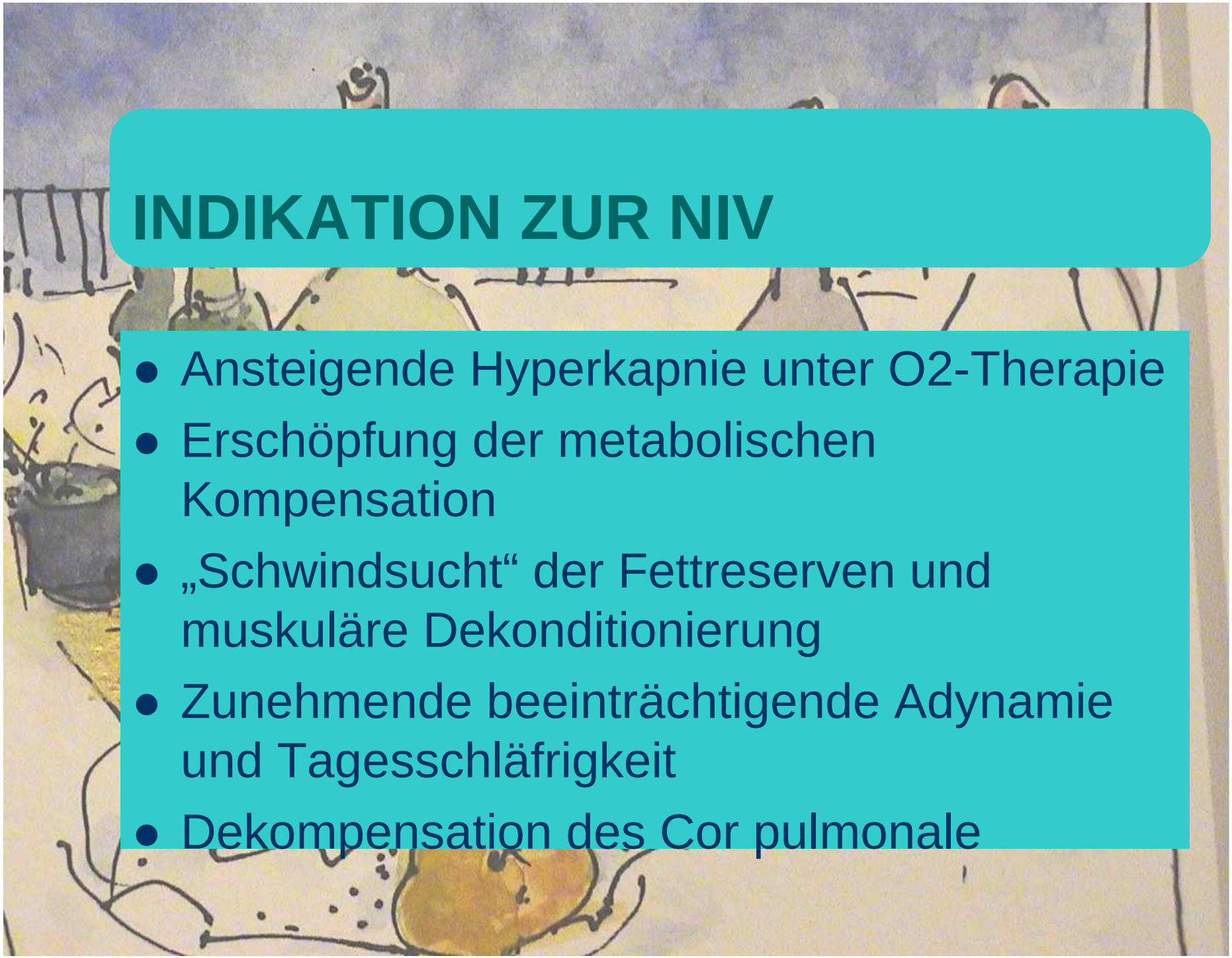
Hirnareale bei Dyspnoe durch CO2-Reiz



Liotti, Mario et al. (2001) Proc. Natl. Acad. Sci. USA 98, 2035-2040

Überdruckbeatmung
Tracheostomie

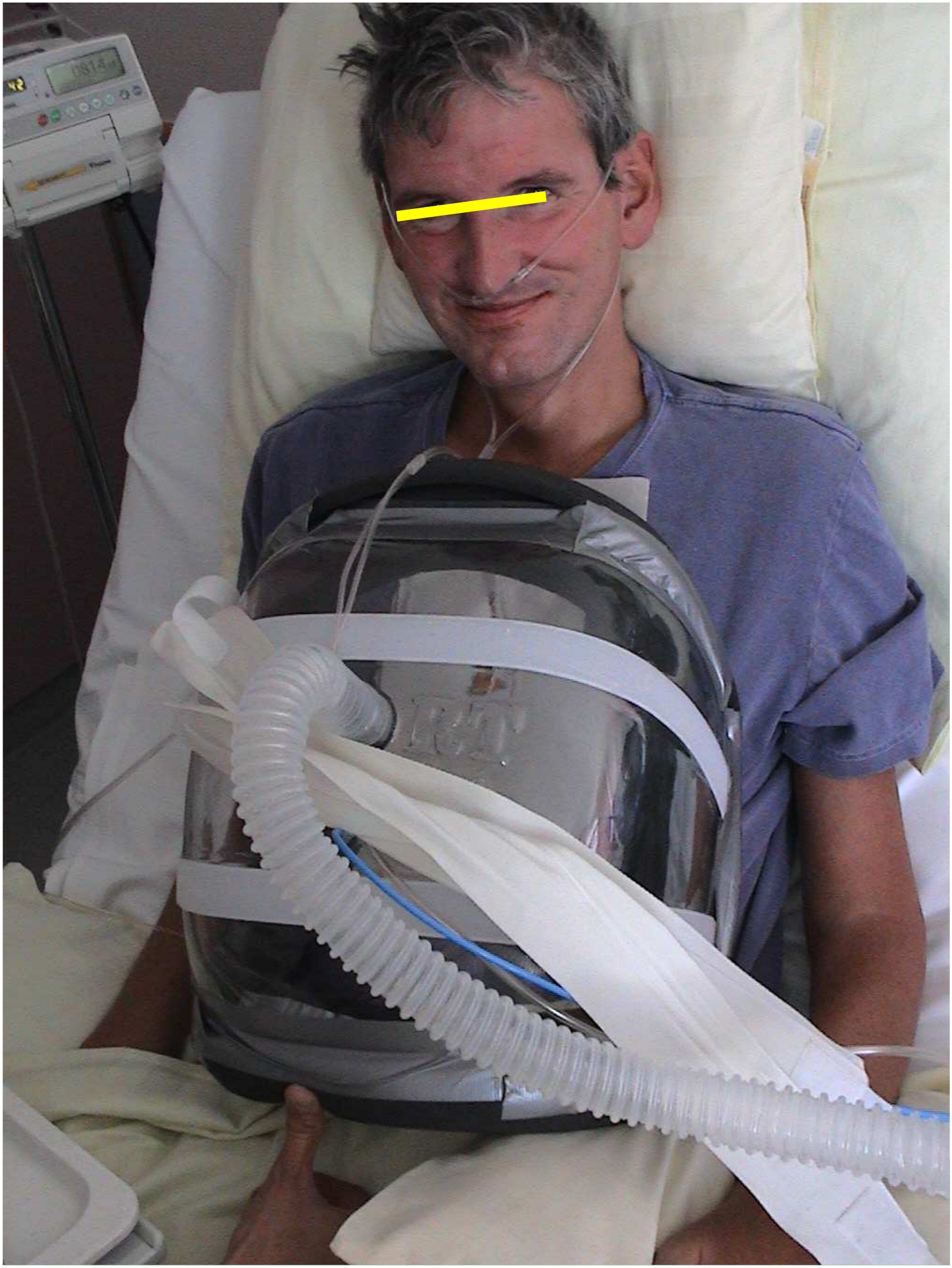




INDIKATION ZUR NIV

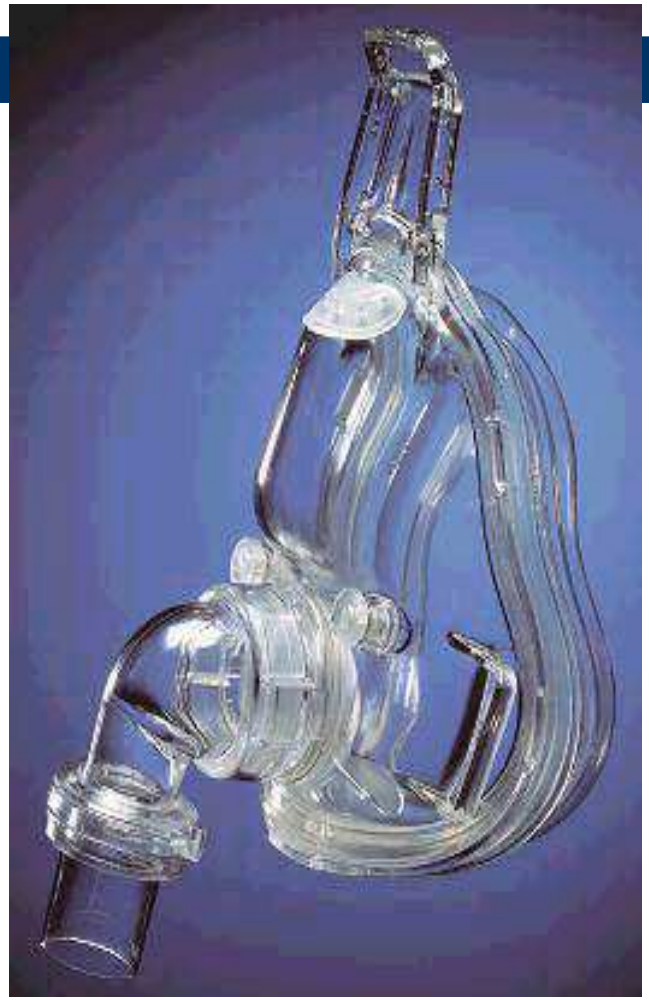
- Ansteigende Hyperkapnie unter O2-Therapie
- Erschöpfung der metabolischen Kompensation
- „Schwindsucht“ der Fettreserven und muskuläre Dekonditionierung
- Zunehmende beeinträchtigende Adynamie und Tagesschläfrigkeit
- Dekompensation des Cor pulmonale

Unterdruckbeatmung

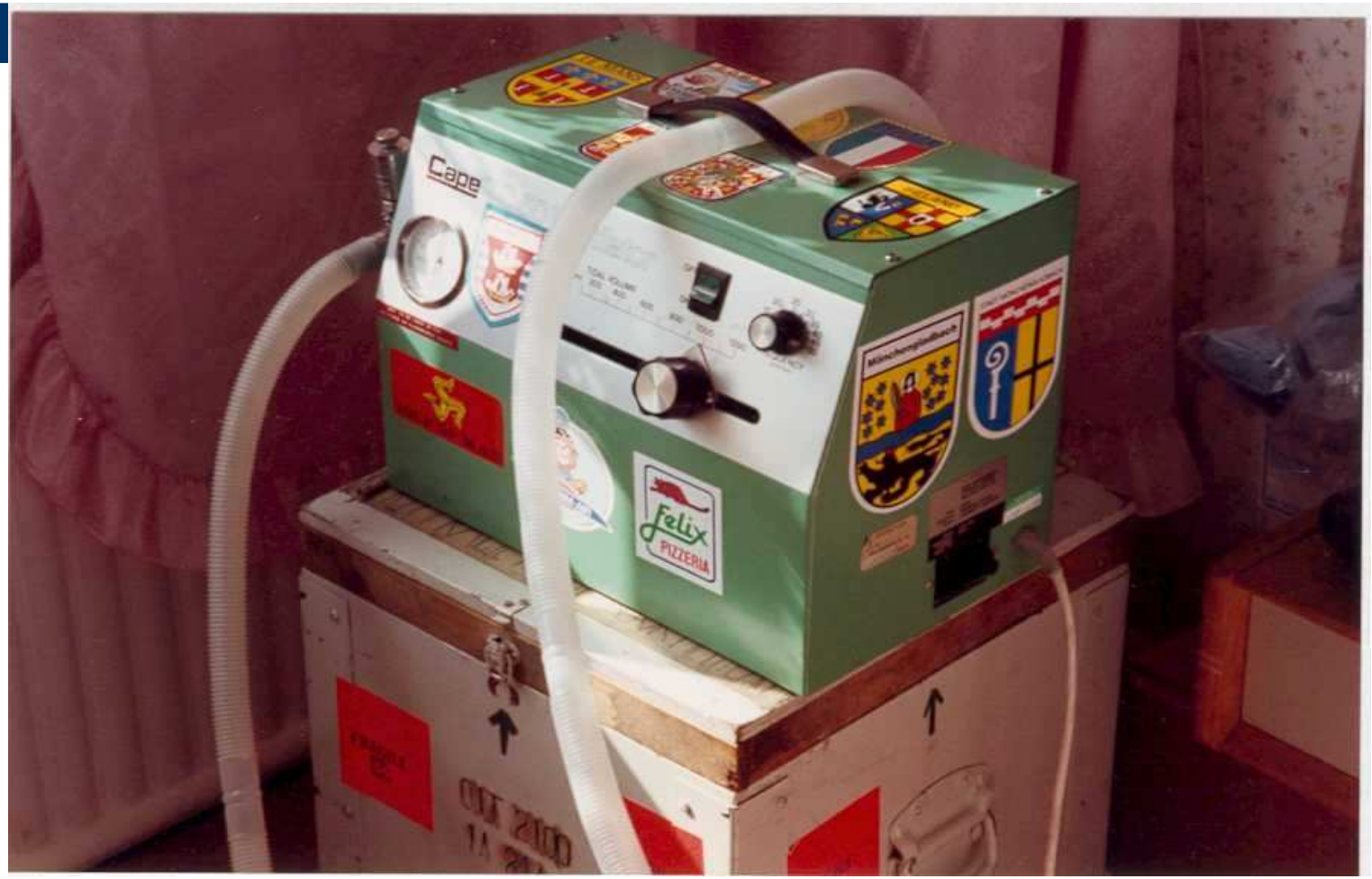


Nicht-invasive Überdruck-Beatmung

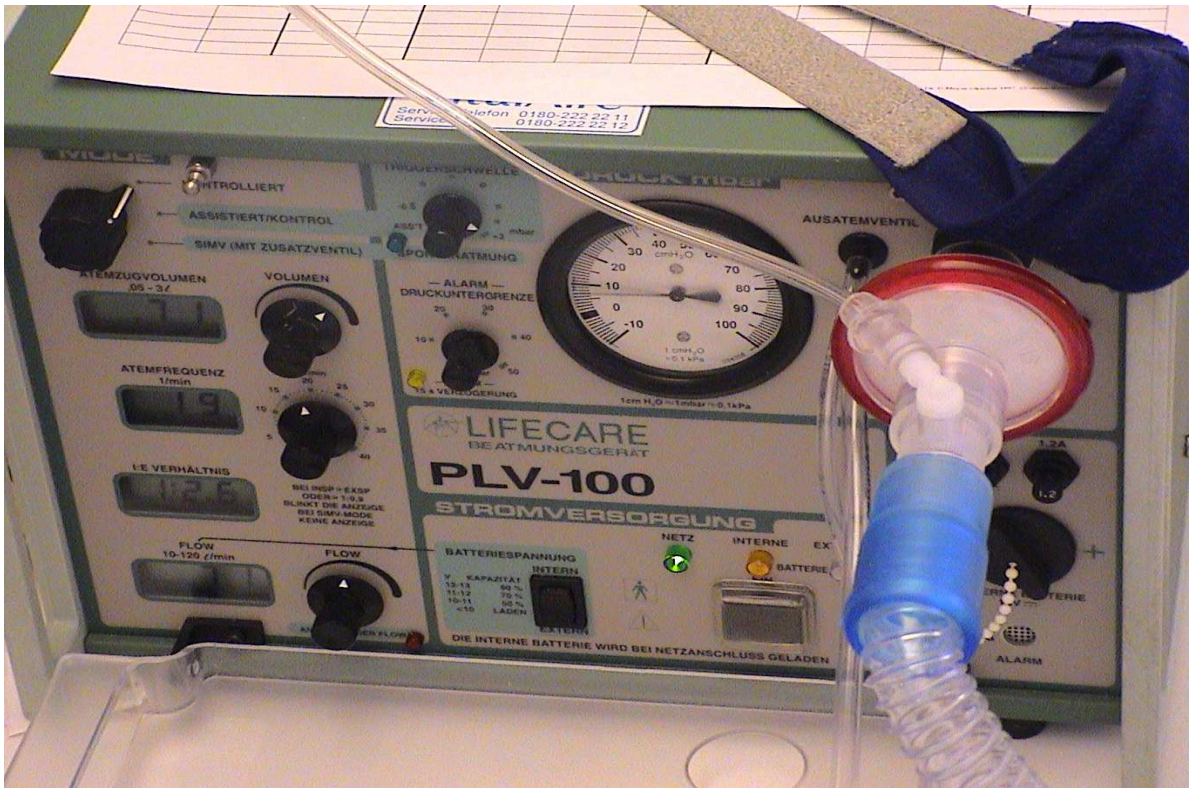
Masken-Systeme



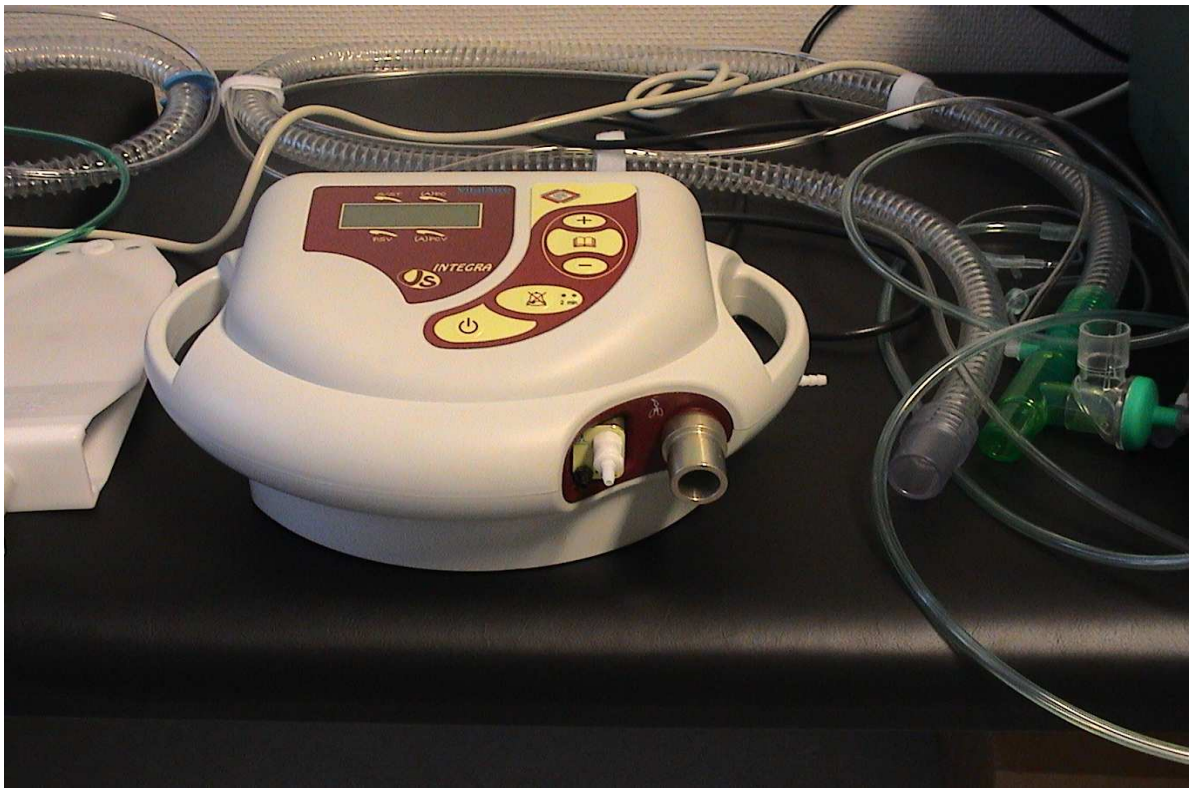
Geschichte und Entwicklung



Überdruckbeatmung
Volumen-Kontrolliert



Überdruckbeatmung
Druck-Kontrolliert



Abgestufte Palette an modernen NIV-Beatmungsgeräten

Moderne Geräte zur Invasiven Heimbeatmung

Sauerstoff-Therapie

Sekret-Problematik

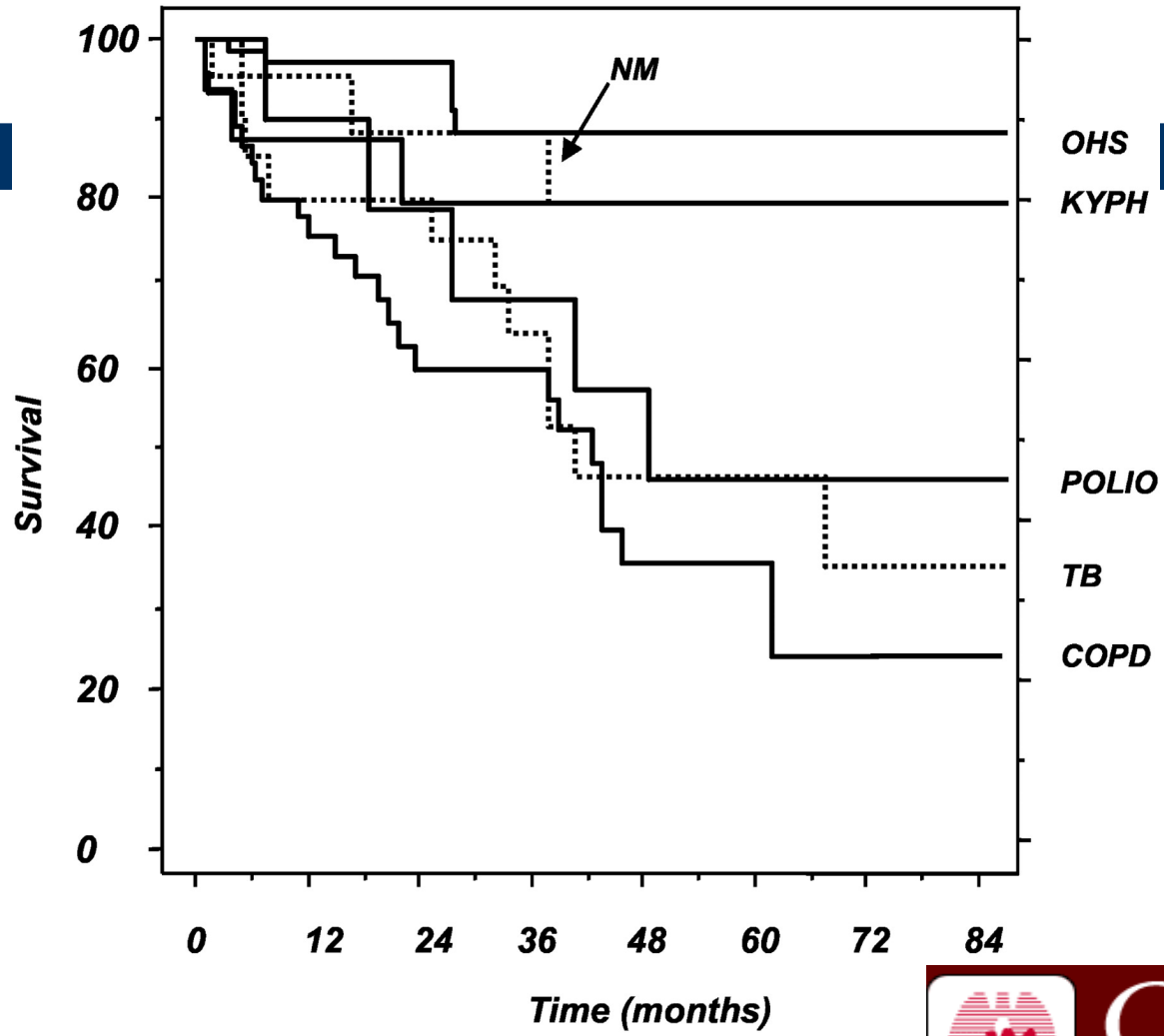
Cough Assist

Probleme

- Akzeptanz durch Patient und dessen Umfeld
  - Grundlegende Bedürfnisse wie Essen, Trinken, Defäkation, Hygiene
  - Ermöglichung von Kommunikation
- Handhabarkeit auch durch „Laien“
- Verläßlichkeit und Robustheit
- Immanente Komplikationen

Was hat der Patient davon?

- Lebensqualität
- Lebensverlängerung
- Mehr Leistungsfähigkeit

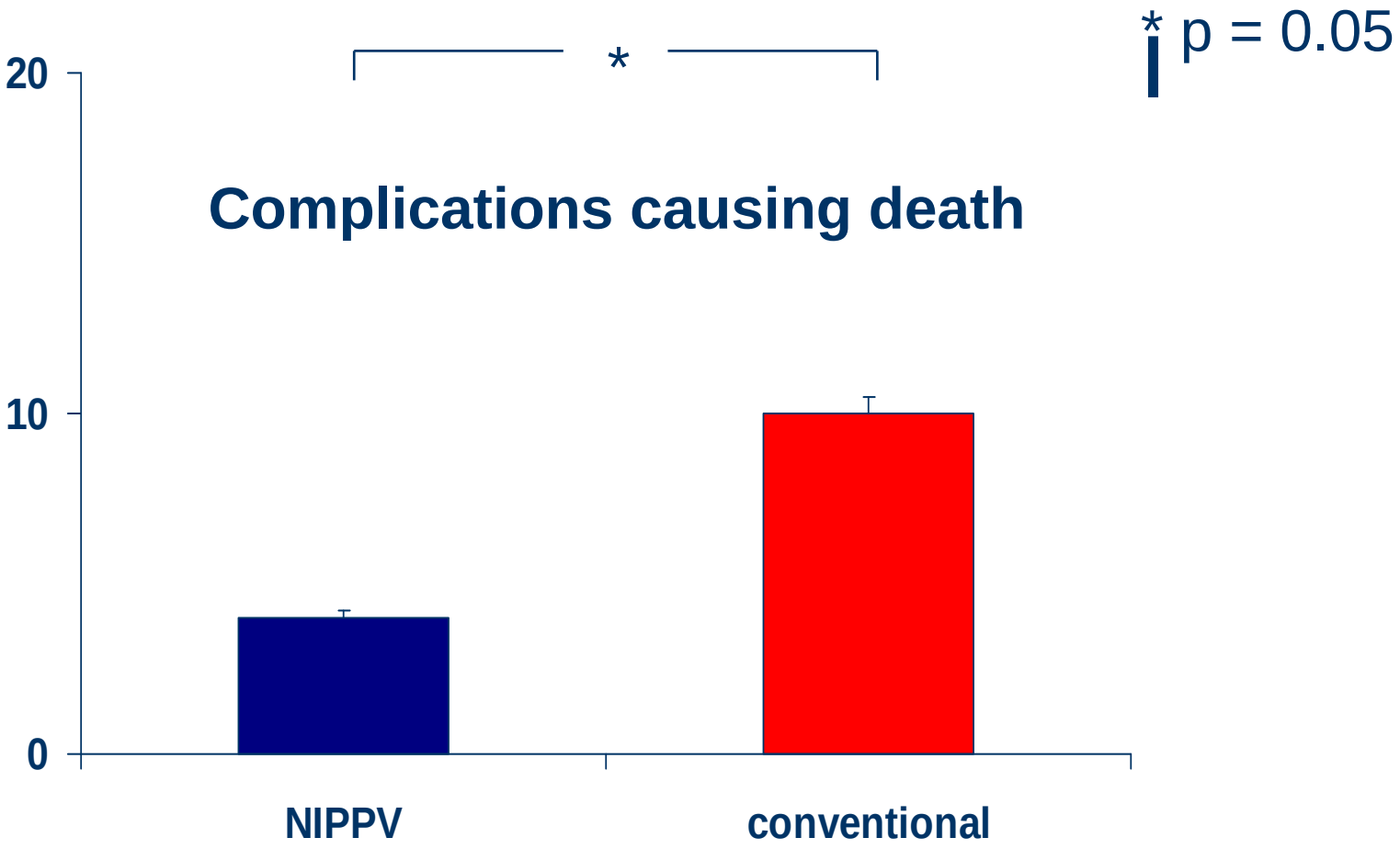


Janssens, J.-P. et al. Chest 2003;123:67-79

CHEST
The Cardiopulmonary and Critical Care Journal

NIV bei ARI - hypoxämisch

Antonelli et al., JAMA 2000, 283:235



Therapieerfolge