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EFFECTS OF CURATIVE TREATMENT EMPHASIZING ENDURANCE TRAINING ON THE PERFORMANCE AND BLOOD PRESSURE OF HYPERTENSIVES AND NORMOTENSIVES

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tontem Training auf Leistungsparameter und Blutdruck bei Hyper-
tonikern und Kreislaufgesunden; 2. Mitteilung: Normwertproblematik
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16. Abstract Blood-pressure behavior of 26 patients with hypertonic blood pressure after exercise, as well as that of 80 healthy subjects (men aged 40-59 years) at the beginning and at the end of a 4-week cure with endurance training is analyzed. The author discusses the problem of normal values of blood pressure after exercise taking into account the blood pressure at the end of the exercise test. Hypertensives showed a lower working capacity than normotensives. In normotensives, however, systolic blood pressure at the end of an exercise correlated well with the working capacity. After the endurance cure submaximal blood pressure was markedly lower in hypertensives with a striking dependance on the level of initial values. Systolic blood pressure at the end of an exercise test was not changed significantly. Most probably it is not possible to overcome this malregulation in hypertensives by endurance training alone. Obviously, this can be achieved only after the additional administration of antihypertensive drugs.

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EFFECTS OF CURATIVE TREATMENT EMPHASIZING ENDURANCE TRAINING ON THE PERFORMANCE AND BLOOD PRESSURE OF HYPERTENSIVES AND NORMOTENSIVES

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<u>Summary</u>   /1207*

The blood-pressure behavior of 26 patients with hypertonic blood pressure following exposure to exercise and that of 80 subjects with normal blood pressure (men aged 40 to 59 years) at the beginning and at the end of a 4-week cure emphasizing endurance training is analyzed. The author discusses the problem of normal blood pressure values following exercise taking into account the exposure values. The hypertensives showed a lower performance capacity than normotensives. In normotensives, however, systolic blood pressure after exposure to exercise correlated positively with their performance capacity. After the endurance cure the submaximal blood pressure in hypertensives showed a remarkable decrease with a striking dependence on the level of initial values. There were no significant changes in the systolic blood pressure following exercise. This malregulation in hypertensives probably cannot be alleviated by endurance training alone. This can obviously be achieved only through additional administration of antihypertensive medications.

Physical exercise in doses stressing endurance is recommended by many authors for treatment of lighter forms of essential hypertonia in stages I and II and of hypertonic regulatory disturbances (10, 11, 17, 46, 48, 56, 59, 63-65, 72, 80, 81, 99, 100 and others). In addition to many short communications involving small numbers of patients, detailed studies citing positive results obtained with larger groups of experimental subjects have been provided by Drews

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*Numbers in the margin indicate pagination in the foreign text.

1

(14, 15), Chomazjuk et al. (10), Schwalb and Behrens (86), Kriz et al. (50, 51), Tittmann et al. (97), Grosse et al. (19) as well as groups working with Chrastek (11, 71), Strauzenberg (95,96) and Priebe (63-65). Reports on results obtained in endurance training cures for hypertonics are relatively sparse (11, 14, 15, 19, 48, 50, 51, 71, 95, 96). In addition to these favorable findings, the references contain also some less optimistic voices (16, 74). After the first part of this study dealt with performance parameters and heart frequency (HF), this second installment will more closely analyze the blood-pressure (RR) behavior in hypertonics and normotensives during the progress of the cure.

Materiel and Method

See 1st installment.

Findings

Next, listed in Table 1, are the RR exposure values determined in normotensives at the outset of the cure as a reference value for subsequent discussion or for comparison with reference data regarding normal values. The term "terminal exposure value" is to be interpreted as meaning the last value measured prior to termination of exposure called for in the case of the test subjects either due to exhaustion or due to having exceeded the age-related so-called terminal HF exposure ($150 \ \text{min}^{-1}$ 40-49 years or $145 \ \text{min}^{-1}$ 50-59). The simultaneously determined effective terminal HF was a median $153.5 \ \text{min}^{-1}$.

Subsequent analysis of the RR behavior will be performed only in the case of certified normotensives (Group I, n=80) and certified hypertonics with actually increased systolic RR exposure at the outset of the cure cycle (Group II, n=26). The "intermediate group" with normal systolic exposure RR (Group III, n=26; for details see 1st installment) was also subjected to computations, but will not /1208 be referred to in this installment to preserve a better clarity of the text.

2

TABLE 1. RR EXPOSURE ON A BICYCLE ERGOMETER (SITTING POSITION, 60 REVOLUTIONS PER MINUTE) FOR THE NORMOTENSIVE MEN IN THE AGE RANGE 40-59 YEARS

	Systolic RR			Diastolic RR			HF	
	x̄	±	s	x̄	±	s	x̄	n
50 Watt	19,8	(149,0)	±1,7(12,5)	11,6	(87,3)	±1,1 (8,1)	99,6	57
100 Watt	23,4	(175,8)	±1,7(13,1)	11,9	(89,4)	±1,4(10,7)	121,0	80
150 Watt	27,1	(203,8)	±2,2(16,3)	12,2	(91,6)	±1,7(12,9)	149,1	64
Ausbelastung	27,4	(205,9)	±2,4(18,4)	12,2	(92,1)	±1,8(13,4)	153,5	78

x = median value; s = standard deviation; n = number of test subjects; RR = blood pressure; HF = heart frequency; Ausbelastung = terminal exposure
RR measuring unit: kPa, in parentheses mmHg

TABLE 2. PERFORMANCE CAPACITY AND HF ON BICYCLE ERGO-METER FOR NORMOTENSIVES (n = 80) AND HYPER-TONICS (n = 26) AT OUTSET OF THE CURE CYCLE

	Normotoniker x̄±s	Hypertoniker x̄±s	P
Wattleistung(W)	190,1 ± 21,7	163,3 ± 21,9	<0,01
Fahrzeit auf dem Ergometer (min)	15,9 ± 3,4	13,6 ± 3,3	<0,001
HF 50 Watt	99,6 ± 10,1	104,9 ± 9,9	<0,05
HF 100 Watt	121,0 ± 11,4	129,0 ± 12,8	<0,01
HF 150 Watt	149,1 ± 12,7	153,9 ± 12,5	ns

x = median values; s = standard deviation; p = error probability in medium value difference; ns = not significant; HF = heart frequency; Normotoniker = normotensives; Hypertoniker = hypertonics; Wattleistung = watt output; Fahrzeit auf dem = duration of exercise on the

Table 2 shows a comparison of the median values of performance and of HF exposurefor both groups at the outset of the cure cycle. The t-test was used for statistical verification of differences. In addition to this mutual group comparison the eventual dependence between performance and HF exposure on the one hand as well as RR exposure on the other hand was checked separately for normotensives and hypertonics. In the case of normotensives it was possible to establish a positive correlation between performance (duration of exercise) and terminal systolic RR exposure ($r = +0.37$, $p < 0.001$) as well as a negative correlation between HF exposure (e.g. 100 W) and terminal systolic RR exposure ($r = -0.40$, $p < 0.001$). On the other hand, there was no correlation between performance and the systolic and diastolic RR at 100 W, and none between HF exposure (100 W) and systolic RR at 100 W. The same result is pointed out

3

once again by group performance comparison in Table 3. Notable
in these values is the indicated inverse (yet not significant)
behavior of the submaximal systolic RR in comparion to the ter-
minal systolic RR exposure. The group of hypertonics showed, on
the whole, similar tendencies, but statistical significance could
not be established either in correlation analysis or in the group
comparison (see Table 3). The varying warm-up exercises of the
patientsshowed in normotensives no effects on the systolic RR ex-
posure: at 100 W the values for all three warm-up exercise groups
were virtually identical.

TABLE 3. SELECTED CIRCULATION PARAMETERS IN EXPOSURE
WITH VARYING PERFORMANCE CAPACITY IN NORMO-
TENSIVES AND HYPERTONICS

Normotoniker (n = 80)	Leistungsfähigkeit		p
	überdurch-schnittlich	unterdurch-schnittlich	
HF 100 Watt	116,3	126,4	< 0,001
Systolischer RR 100 Watt	23,0 (173,2)	23,8 (178,9)	ns
Systolischer Ausbelastungs-RR	28,0 (210,7)	26,6 (199,9)	< 0,01
Hypertoniker (n = 26)			
HF 100 Watt	130,8	137,1	< 0,001
Systolischer RR 100 Watt	27,9 (209,4)	28,5 (214,1)	ns
Systolischer Ausbelastungs-RR	31,6 (237,5)	30,6 (230,2)	ns

Normotoniker = normotensives; Leistungsfaehigkeit =
performance capacity; ueberdurchschnittlich = high
average; unterdurchschnittlich = low average; p =
error probabilit" in medium value difference;
Systolischer = systolic; Ausbelastung-RR = terminal
RR exposure; RR measuring unit: kPa, in paren mm Hg

Following the endurance training cure cycle the RR at rest in
the morning in normotensives showed hardly any change; systolic
from 15.4 kPa (116 rmHg) to 15.0 kPa (113 mmHg) ($p < 0.01$), diastolic
remained constant at 10.1 kPa (76 mmHg). In hypertonics these
values went back from systolic 17.7 kPa (133 mmHg) to 16.5 kPa
(124 mmHg) ($p < 0.001$), diastolic from 11.7 kPa (88 mmHg) to 10.6 kPa

(80 mmHg) (p<0.001). The RR measured in hypertonics at the outset
of the cure cycle during medical admittance examinations was at a
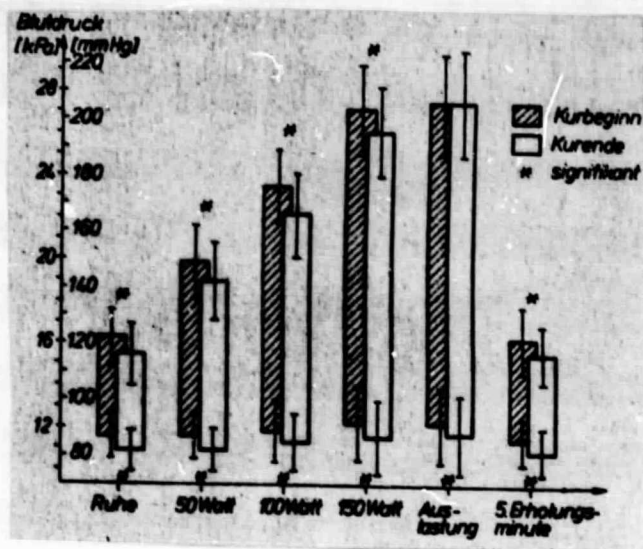median of 21/13 kPa (157/98 mmHg).

Fig. 1. Blood pressure in normotensives (n = 80) during
ergometry during the course of curative cycle

Blutdruck = blood pressure; Kurbeginn = start of cure
cycle; Kurende = end of cure cycle; Ruhe = at rest;
Auslastung = exposure; 5. Erholungsminute = 5th minute
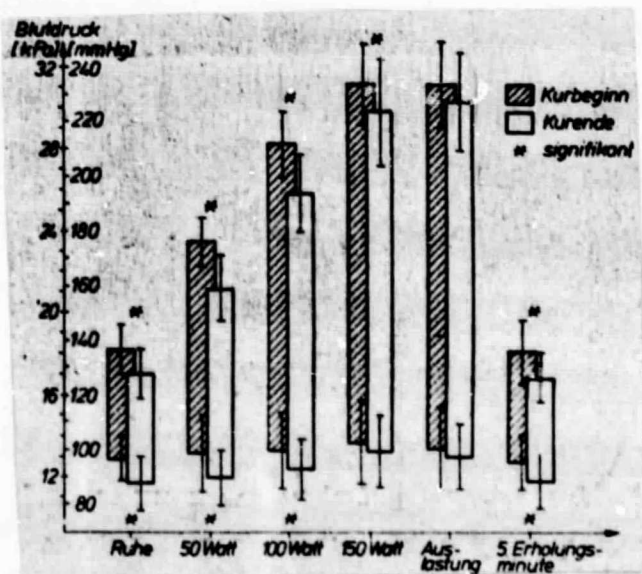of recovery

Fig. 2. Blood pressure in hypertonics (n = 26) during
ergometry in the course of the curative cycle

Key = same as for Fig. 1 above.

5

Figure 1 shows the dynamics of RR behavior during ergometry (at rest, during exposure, during recovery) in the course of the curative cycle for normotensives, figure 2 shows it for hypertonics. At identical watt stages, systolic decrease in normotensives amounted to 0.9 to 1.3 kPa (7-10 mmHg), diastolic 0.7 to 0.8 kPa (5-6 mmHg). On the other hand, these decreases up to 100 W for hypertonics amounted to 2.4 kPa (18 mmHg) for systolic and 0.9 to 1.1 kPa (7-8 mmHg) for diastolic, decreasing with increasing exposure. The initially exceeded submaximal median values returned /1209 to normal. With the exception of terminal systolic RR exposure in both groups and diastolic RR at 150 W and terminal exposure of hypertonics, all differences are significant. From among the 26 hypertonics, 19 showed a systolic and 16 a diastolic RR decrease at all watt stages. Increase of RR values in pathological or in the borderline range was registered in the following patients: systolic RR exposure - 4 patients, diastolic RR at rest - 6 patients (simultaneously with diastolic RR exposure once and with systolic RR at rest twice).

The extent of decrease in submaximal systolic RR was positively correlated with the initial value at the corresponding stage, closer in hypertonics (r = +0.56, p<0.01) than in normotensives (r = +0.36, p<0.01) (computed for the 100 W stage). As HF as well as RR kept changing through the endurance training curative cycle, checks were also made as to what the systolic RR behavior was at the outset and at the end of the curative cycle always at identical HF and whether any differences could be detected between the hypertonics and normotensives. To that end we constructed regression curves between both values approximatively from the median values of the ergometric data (Fig. 3). Computation of regression equations was dispensed with, as regressions in the upper ranges did not appear to be linear. The curves flatten out, meaning that HF increases faster than systolic RR with higher exposure.

As described in the 1st installment, we proceeded to separate from this phase all patients who were newly administered antihypertensive medicinal preparations during the curative cycle or those
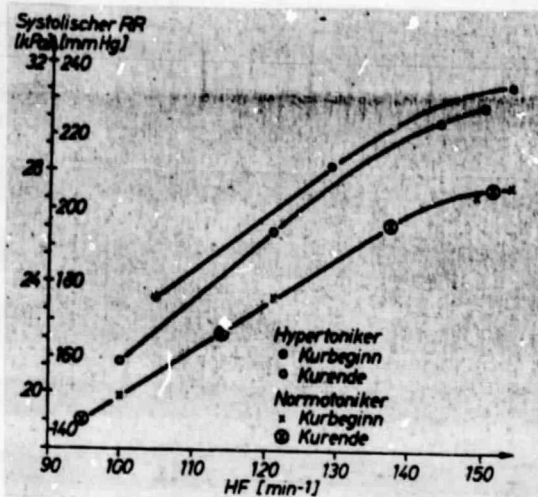
Fig. 3. Relationship between systolic blood pressure and heart frequency during exposure at the outset and end of the curative cycle in normotensives and hypertonics

Systolischer RR = systolic blood pressure; Hypertoniker = hypertonics; Kurbegin = start of curative cycle; Kurenede = end of curative cycle; Normotoniker = normotensives

whose dosage had undergone changes. However, due to conspicuities in terminal systolic RR exposure we still deem it of interest to report on the progress in values of systolic RR exposure of those male patients of the same age group with essential hypertonia in stages I and II (n = 8) who received no prior doses of medicines, for whom -- in addition to circulatory endurance training and other curative measures -- it was deemed necessary to establish a new regime of administration of antihypertensive medical preparations (6-times Rauwolfia alcaloids (Rauwocomb , registered trademark), 2-times diisopropyl amine (Disotat, registered trademark), 1-time additionally hydrochlorothiazide (Disalunil, registered trademark) always in small to medium dosage). We are, of course, aware of the fact that this type of group formation makes comparative statements subject to dispute and that such statements must be made with due reservations. These 8 patients showed the following dynamics of median values of the systolic RR exposure in the course of the curative cycle:

	Kurbeginn [kPa] [mmHg]	Kurende [kPa] [mmHg]	p
Syst. RR 50 Watt	25,7 (193,5)	21,0 (158,1)	<0,01
Syst. RR 100 Watt	29,8 (223,8)	24,8 (186,8)	<0,001
Syst. RR bei Ausbelastung	32,7 (245,5)	30,0 (225,9)	<0,01
HF bei Ausbelastung	134,9 min⁻¹	135,3 min⁻¹	n. s.

Kurbegin = start of curative cycle; Kurenede = end of curative cycle; p = error probability in median value difference; bei Ausbelastung = during terminal exposure

Discussion

Normal value specifications for RR exposure in steady-state tests are available in technical literature only for the submaximal range (60, 69). The systolic RR of our normotensives (see table 1) corresponds with them very well while the values quoted for diastolic RR are somewhat higher. We deem it of importance that RR be tested also at higher exposure stages up to the point of terminal exposure even though there still are some uncertainties in weighing of these values. The available findings show that a terminal systolic exposure of 26.6 kPa (200 mmHg) represents in average the norm, with 2-s scattering up to 30.6-31.9 kPa (230-240 mmHg), i.e., values which in clinical practice are held by consensus to represent the terminal criterion. Similar or even higher values were found by other authors in endurance runs (1) or in ergometric bicycle Vita-maxima tests (quoted in 57 and 99). A sharply divided line between normotensives and hypertonics cannot be expected to be encountered as transitions are smooth (69). With the use of normal systolic values in terminal exposure and performance capability or HF we are of the opinion that in some cases the clarity of the dividing line can be increased while avoiding eventual faulty assessment of the problem which is of such importance to prognostic evaluations and monitoring or treatment consequences (whereby there still are uncertainties regarding the significance of the isolated exposure hypertonia (4, 6)):

1. Falsely positive findings in submaximal stages with poor economy of circulation, high HF exposure and largely limited performance capability, which probably for their major part could be classified under the term "hyperkinetic cardiac syndrome" with increased submaximal cardiac minute volume, whereby the principal delineation of this syndrome from "hypertonic regulatory disturbance" and from "high-pressure exposure" in technical literature is not unequivocal (20, 69). The terminal systolic RR exposure is hereby below 30.6 kPa (230 mmHg). The supposed exposure hypertonia "disappears" in these cases with simple economy of circulation. Without any doubt, some of what is involved is a matter of definition.

At the very least, what is involved are actually two differing /1210
haemodynamic situations.

2. Falsely negative findings at submaximal stages when at
high performance capacity with economic circulatory regulation
(which can also occur with lighter forms of hypertonia) excessive
systolic RR values are reached only at higher watt ranges. Hereby
the terminal RR exposure of over 30.6 (-31.9) kPa (230-240 mmHg)
is a decisive criterion. This applies to the average populace.
Other conditions apply to sportsmen engaging in endurance per-
formances (see below). Diastolic RR exposure will not be dealt
with in this context due to the known metrological uncertainties.

As can be seen from table 2, our hypertonics, in agreement
with other reports (49, 54, 55, 77, 78, 85) show a significantly
smaller performance capacity with higher submaximal HF than is the
case with normotensives. In relation to terminal systolic RR ex-
posure the conditions in the case of normotensives are reversed:
performance capacity significantly increases with higher RR while
the submaximal HF exposure decreases. Exclusive consideration of
the submaximal systolic RR, wherein we found no significant cor-
relations, led other authors to diametrically opposite conclusions
(55, 85). Others have repeatedly maintained that specifically
hypotonic systolic exposure values are associated with a limited
performance range (26, 47, 57, 68). At an overall lower level of
performance and RR our results obtained with normotensives do in
a way correspond with findings obtained many times with endurance
sportsmen (26, 33, 35, 36, 42,55, 57, 79). A high terminal systolic
RR exposure (maximum data up to 38.6 kPa (290 mmHg)) is regarded
here as a beneficial volume reaction and as a sign of a remarkable
contractability of the heart, as there is a positive correlation
with heart size or performance capacity (33, 36, 42).

Contrary to other studies (40, 76), we found no correlations
between performance capacity and diastolic RR exposure in normo-
tensives.

9

Following the endurance training curative cycle the values at rest, submaximal exposure and recovery RR in most normotensives decreased significantly, however only slightly. As can be seen from figure 3, the slight decrease in systolic RR exposure is obviously due to a simple improvement in circulatory economy, as in the same relation it is parallel to a decrease in HF. The regression curves between HF and systolic RR at curative cycle outset and end show precise coincidence, i.e., at even HF the same systolic RR is attained prior to and after endurance training.

In the case of hypertonics it must be stressed that median values at rest were quite low already at the beginning of the curative cycle, as only light forms of the disease or exposure hypertonia were concerned, which in part were also treated by anti-hypertensive medical preparations. More serious forms of hypertonia must achieve a decrease in pressure through administration of medicines a priori before endurance training can be engaged in without risks.

Contrary to the normotensives, systolic RR decreases in hypertonics were substantial, diastolic only slightly more pronounced. The submaximal median values returned to normal. RR decrease could be documented for the majority of the patients, an increase in the pathological or borderline range only in exceptional cases. At 2.4 kPa (18 mmHg) the systolic RR exposure decrease (up to 100 W) did occur in ranges similar to those reported for other endurance training curative cycles (15, 50, 96). A (smaller) RR decrease was recorded (figure 3) not only at identical watt stages, but also at an identical HF. Thus, in the case of hypertonics the RR reduction effect exceeds the confines of a simple improved economy of circulation, even though the latter obviously plays an important role in the effective mechanism of endurance training (enhancement of the often present circulatory hyperkinesis (63, 64). Technical literature in addition offers other theories in regards to the effective mechanism: Reduction of peripheral resistance through dilation of vessels in the musculature (32, 99), displacements in neurovegetative equilibrium in favor of the trophotropic side (11, 63, 64, 86, 100), and psychical relaxation (11).

At this point we must again, as we did in the 1st installment, point out that due to the lack of a control group not exposed to endurance training, the extent to which other curative effects may have favorably affected the results obtained with the RR of our patients, must obviously remain moot.

Effects of the decrease in submaximal systolic RR were positively correlated in both the hypertonics and, to a lesser degree, in normotensives with the initial value. Similar observations (48, 64) or lack of effects on the normotonic as opposed to the hypertonic initial state (14, 15, 95, 96) have already been reported by other authors.

How important are in this respect tests in higher performance ranges is pointed up by the fact that in our hypertonics (as well as in the normotensives) the terminal systolic RR exposure after the curative endurance training cycle showed no significant changes in comparison to the submaximal values at identical watt stages. The slight decrease in RR at 150 W in comparison to the lower watt stages must be explained in the same way, as this stage represented for many patients the maximum performance. Herein the hardly changed terminal RR exposure was of course first reached at a higher stress level with, however, almost identical HF values. Thus, this mal-regulation can probably not be eliminated through endurance training alone which, no doubt, serves to limit the value of this therapy to a certain degree. We found no similar reports in the available technical literature. The latter never offered a description of the terminal RR exposure -- with the exception of Strauzenberg et al. (95, 96) who, contrary to the results obtained by us, determined in hypertonics after a curative endurance training cycle considerable and significant decreases in the "terminal RR exposure value".

From the viewpoint of the just described findings, the results obtained with additional administration of antihypertensive medical preparations assume a special significance. As can be seen (with the proviso of the above described methodology), it produces not only an increase in the level of RR decrease at submaximal levels

in the course of the curative cycle. The terminal systolic RR ex-
posure also decreases significantly by 2.7 kPa (20 mmHg), yet it
still lags behind the findings made at the submaximal levels (ap-
proximately 4.7 kPa (35 mmHg)). Continued systematic investigations
in this field seem advisable.

In view of our findings we consider a training treatment stress-
ing endurance to be on the whole a suitable measure for treatment,
particularly that of light or boundary-value hypertonias which, ac-
cording to the present state-of-the-art still do not call for any
administration of medicinal preparations. Nevertheless, expectations
must not be set too high. We agree with Schwalb and Behrens (86) as
well as with Tittmann et al. (97) that in many cases -- particularly
at higher RR values -- training must definitely be accompanied by
additional treatment using administration of medical preparations.
This decreases the risk of extreme exceeding of RR through bodily
stress due to endurance training and obviously promotes also a de-
crease in RR exposure. The effects of targeted physical training
on the overall progress of essential hypertonia according to all
available data cannot still be assessed with certainty (64, 65, 86).
Reaching of that goal calls for continued investigations.

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