manner by the anti-BKN (see figure 1). Nevertheless, 3 rabbits treated (1 in group 1 and 2 in group 3) did not give birth. This could be caused by: absence of fertilization; defective implantation of blastocyst; or reabsorbment of embryos already nidated. These last 2 hypotheses are confirmed by the experience of Krishnan[10] and Daniel[11], respectively. In analyzing the prenatal mortality in the different groups of treatment, it is obvious that the major incidence occurs in the rabbits treated in groups 2 and 4.

The mean of the weight of the stillborn in comparison with the weight of the live young from the same rabbits after 3 months, is constantly lower (see figure 2). This phenomenon is more evident in groups 2 and 4, and it could be related to the arrest of growth resulting in uterine death of the foetus. Above all the appearance of the stillborn leads one to believe that death occurred recently before birth. Therefore the reduction in weight could be due to the under-development of the foetus. Moreover, the stillborn young under the macroscopic examination, seemed to be normal and properly formed; it therefore seems to exclude a teratogenic activity of the anti-BKN 'in vivo'. Our results did not show confirmation of defective implantation or reabsorption of the embryos produced from the anti-BKN administration; but, on the other hand, the effect on the prenatal mortality and on the weight of the young at birth was evident. It has been demonstrated that the period of greatest biological effect of anti-BKN corresponds with the period of maximum production of blastokinin by the endometrium, i.e., about the 4th–5th day following coitus[15]. This confirms the hypothesis of the importance of the production and the presence of the blastokinin in the uterus at the moment of blastocyst nidation. Nevertheless, it is not clear how the activity of the anti-BKN can produce a high prenatal mortality. We way presume that it can neutralise the blastokinin in its biological role, connecting it, in vivo, with the development of the placenta.

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The effect of sleep deprivation upon variations in heart rate and respiration rate

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Summary. Variations in heart rate and respiration rate were found to be responsive to total sleep deprivation, particularly under experimental conditions more realistic to everyday life.

From recent reviews[2–4] and studies[5–7] on the effects of total sleep deprivation (TSD) in man, the major findings appear to be amongst behavioural, performance and EEG parameters rather than with physiological measures of somatic functioning. Although this suggests that human sleep may be more oriented towards the brain than to the body, there are methodological factors to consider. Firstly, because of experimental control, subjects are usually confined to a laboratory, and apart from the TSD, lead a restricted regime unlike a normal everyday existence. If human sleep aids restitution from wakefulness, then this regime may have a reduced effect than otherwise might be expected, particularly as it has been hypothesised[8,9] that a high waking visual load might potentiate TSD effects. Secondly, certain physiological measures may not be sensitive enough to TSD. Measures taken during any physiological investigation ideally need to have conceptual validity to the phenomenon under examination. Respiration is apparently[10,11] very sensitive to changes in consciousness. This factor together with: a) the apparent[12–14] interplay between cardiac regularity mechanisms, cardiac output and levels of attention, b) reports[15–17] that attention levels affect sinus arrhythmia, with respiration playing a major intermediary role, all suggest that although mean levels of heart rate (HR) and respiration rate (RR) are not sensitive to TSD[2–7], HR variation (HRv) and RR variation (RRv) may be so. The only study[18] to assess HRv found significant, but undetailed, changes. RRv has not been used in TSD studies.

Method. 6 healthy young males were paid to stay awake for 62 h, on 2 occasions. Both occasions were laboratory centred, with 1 having a high visual perceptual load and the other being a control condition containing a low load, more typical of other TSD studies. These conditions are detailed elsewhere[8]. Subjects were TSD'd in 2 groups of 3 with one group undergoing high load-low load, and the other group the reverse order. 2 weeks elapsed between conditions. 3 baseline days preceded each TSD and 2 recovery days followed. Subjects slept in a sleep laboratory on these 5 nights to ensure no additional sleep loss. Using EKG chest electrodes and nasal thermistor, HR

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Summary of analysis of variance findings (1 tail significance levels)

	Sleep deprivation				Recovery days			
	HR	HRv	RR	RRv	HR	HRv	RR	RRv
Between-days	NS	0.001	NS	0.01	NS	NS	NS	NS
Between-times	NS	NS	NS	NS	NS	NS	NS	NS
Between-conditions	NS	0.05	NS	NS	NS	NS	NS	NS
Days-×-times interaction	NS	0.001	NS	NS	NS	NS	NS	NS
Days-×-conditions interaction	NS	0.01	NS	0.01	NS	NS	NS	NS
Times-×-conditions interaction	NS	0.01	NS	NS	NS	NS	NS	NS

HR, heart rate; HRv, heart rate variation; RR, respiration rate; RRv, respiration rate variation; NS, not significant.

and RR were recorded on a FM tape-recorder for 5-min epochs at: 04.00, 10.00, 16.00 and 22.00 h throughout TSD, and at these times where relevant during baseline and recovery days. The 04.00 h baseline data were collected separately, with subjects awake, on 2 occasions following the completion of the 2 TSD conditions. This avoided any sleep disturbance and carry-over effects during the conditions. Subjects were seated but were not passive during measurement as they performed 20 min of simple tracking tasks, which acted as measurement control. Unknown to the subjects, the HR and RR data were collected from min 14–18 inclusive. The laboratory was maintained at a constant and comfortable temperature and humidity. Subjects ate a balanced diet 2 h after each measurement. Calorific intake was 2600 Cal/24 h. Between measurement, subjects were exposed to either high or low visual loads[8]. Exercise was balanced for both conditions. Subjects were continuously observed during TSD to ensure no naps were taken.

Beat to beat intervals for HRv and inspiration to inspiration intervals for RRv were digitized via a Schmidt trigger. For each subject epoch the mean and standard deviation of the first 500 HR intervals and the first 50 RR intervals were computed. These results were converted into rates. The standard deviation was taken as the index of variability. For each subject, averaged data for each of the baseline times were subtracted respectively from each of the TSD and recovery day scores. 3-way analyses of variance were performed on group data as shown in the table.

Results. From figures 1 and 2 and the table it can be seen that there were no significant changes with: a) RR and HR means, b) HRv and RRv for recovery days. For TSD there were significant between-day effects for HRv and RRv and a between-condition effect for HRv. The significant days-×-times interaction for HRv can be seen in figure 1 with the differences in time effect becoming larger with progressive TSD during both conditions. The significant days-×-conditions interaction for both RRv and HRv can be seen in figures 1 and 2, with a progressively greater parting of the 2 conditions over TSD. An interesting times-×-conditions interaction for HRv is reflected in a larger increase in HRv during high load TSD than for low load.

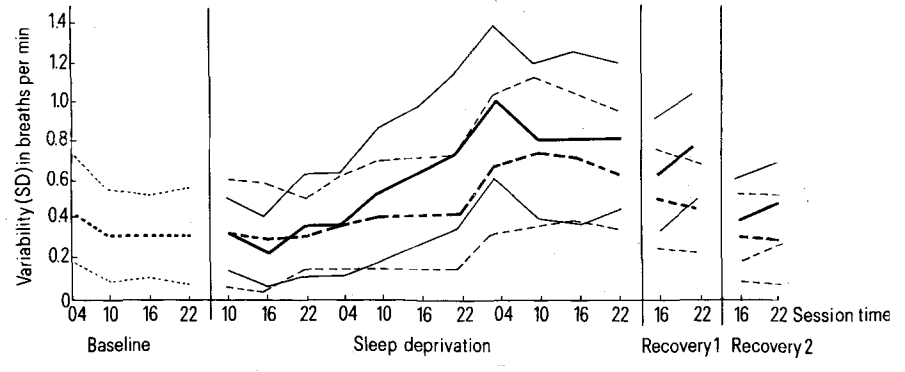

Fig. 1. Changes in respiration rate variation. High load group: ——, mean, and —— SD of the variability. Low load group: ---, mean, and --- SD of the variability.

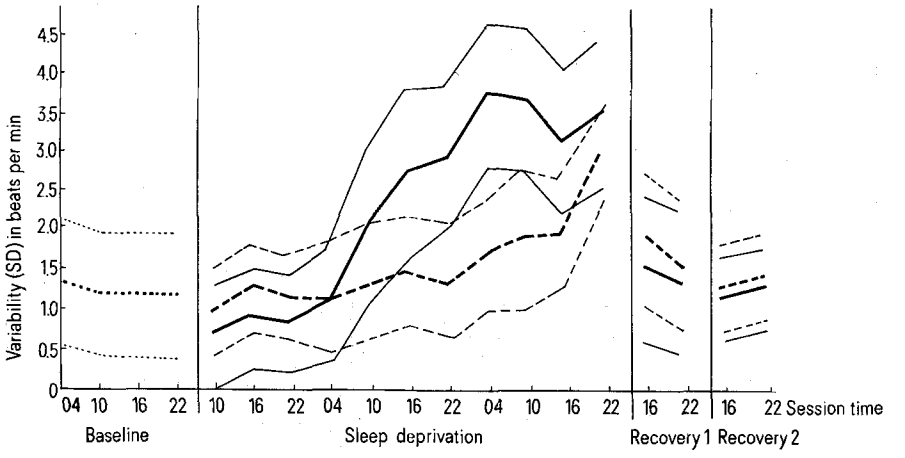

Fig. 2. Changes in heart rate variation. High load group: ——, mean, and—— SD of the variability. Low load group: ---, mean, and --- SD of the variability.

Discussion. From the results it appears that although mean RR and HR showed no significant changes HRv and RRv are sensitive, with HRv being more responsive and also showing a between-conditions effect. Seemingly, the high visual load condition did potentiate the effects of TSD, indicating that experimental conditions of TSD do play a major role. Correlating HRv and RRv scores for each subject over both TSD conditions produced an overall average product moment correlation of 0.78. This 61% common variance indicated that the 2 variables are significantly correlated. Because respiratory sinus arrhythmia (RSA) is a non-linear, respiratory depth and frequency dependent phenomenon[19,20] varying in phase and magnitude, and as RR was allowed to vary, a more detailed partialling out of a RSA component from the HRv data was not possible.

Circadian rhythms in the data were not statistically evident. However, as there were only 4 measures per day, with pooling of data, and a possible circadian rhythm disruption owing to TSD, such an effect might not be obvious.

More sophisticated[15,16] HRv and RRv indices could have been employed but the present method, although simple, was apaprently effective. From observing both subjects and raw data it appears that the RRv increases were often due to increases in yawning and sighing, etc.

The exact role of CNS fatigue and any CNS impairment to HR and RR control cannot be established here, however these subjects did show increasing amounts of behavioural fatigue, especially during the high load condition. These effects are detailed elsewhere[21].

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Changes in Z-disc width of vertebrate skeletal muscle following tenotomy[1]

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Summary. Results of unilateral Achilles tenotomy on male rats, after 2–6 weeks, showed conclusively that the Z-lines of tenotomized muscles are significantly wider than those of control, nontenotomized muscles.

The Z-line of vertebrate skeletal muscle is known to become morphologically altered (rod or nemaline bodies, streams, etc.) in a variety of pathological conditions[3,4] as well as under conditions of hypertrophy[5,6] and tenotomy[7,8]. Figure 1 shows the typical streams and rod bodies following tenotomy. Also Fujisawa[9] found streams in otherwise normal aged rats. He noted that the Z-lines in those affected fibres appear wider (up to 120 nm) than in non-streaming fibres (up to 92 nm). Although the exact function and chemical nature of the Z-discs are not known, it has been suggested that the structural proliferation observed is the first step in the normal development of new sarcomeres[5,10,11].

In the specific case of tenotomy, if one assumes that the stimulus, perhaps a sudden release of resting muscle tension, induces a 'proliferative' response in all the Z-lines of the affected muscle, then this should be detectable by measurement of Z-line width in areas of the muscle which do not exhibit the more obvious and familiar Z-line abnormalities. The following study, therefore, was designed to answer the simple question: Are the Z-lines of tenotomized muscle, apart from those showing rod formation or streaming, thicker as compared to the Z-lines of a comparable, but non-tenotomized muscle?

Materials and methods. Following the method described by Shafiq et al.[7], Achilles tenotomy, including removal of a 2–3 mm segment of tendon, was performed on 1 leg of 150–300 g male albino rats, with the unoperated egl serving as the control. Following a period of 2–6 weeks, the animals were sacrificed by decapitation and the soleus muscles excised and placed in ice cold buffer (0.1 M KCl, 1 mM $MgCl_2$, 5 mM EGTA, 5 mM sodium pyrophosphate, pH 6.8). Under the dissecting microscope, fibres were dissected free, tied to 3 cm fragments of wooden applicator sticks at approximately rest length, and then placed at 4 °C in 4% glutaraldehyde in buffer (7.5×10^{-2} M KCl, 7.5×10^{-4} M $MgCl_2$, 7.5×10^{-3} M Na_2HPO_4, 7.5×10^{-3} M KH_2PO_4, pH 7.0). The samples were then post-fixed in 1% OsO_4 for 1 h, dehydrated in a graded series of ethanol and embedded in DDSA/araldite. Longitudinal thin sections were cut at 60–70 nm on a Porter-Blum MT 2 microtome, and mounted on 200 mesh copper grids. The sections were then stained with 1% PTA, 10% uranyl acetate, and Reynold's lead citrate and examined with a Philips 300 electron microscope. Electron micrographs were taken at the same magnification of both the control and experimental soleus muscles, care being taken to avoid areas in the tenotomized muscle that abounded with the rod shaped and streaming Z-structures.

Direct measurement were taken (independently, by each author) from 8×10 inch enlargements of the various plates using a metric ruler for sarcomere and A-band length, and a desk-top magnifier containing a reticle with

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