CAD, and serum calcium and phosphorus had no significant association with the angiographic severity of CAD.

Narang et al,[6] showed that serum phosphorus had an independent positive association with the angiographic severity of CAD. In this study, we could not measure the amount of calcium deposition in atherosclerotic plaques of patients with CAD, but regarding the unique concentrations of the total serum calcium and phosphorus between 2 groups of patients with and without CAD, our data suggests that the total serum calcium and phosphorus had no relation with IHD.

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Blood and saliva lactate levels during recovery from supramaximal exercise

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Lactate is now recognized to be an important source of energy for skeletal muscle metabolism, measuring blood lactate concentration (samples) provides information not only regarding changes in glycolysis, but also on anaerobic work capacity. Blood sampling involves blood loss, emotional stress and discomfort, whereas saliva is easy to collect and non-invasive. Several authors have described the possibility of evaluating the changes produced in the composition of saliva in response to exercise as a non-invasive method of determining lactate threshold.[1,2] There is some research, which shows parallelism between the saliva and blood lactate response during incremental exercise.[2] The aim of this study is to investigate whether there is a relation between lactate increase of blood and saliva, during seated recovery after supramaximal exercise.

A group of 10 male athletes, practicing an average of 6 hours of sports activities per week, and with an acceptable level of physical fitness, agreed to participate in the study after the objectives of the investigation and the protocol to be followed was explained to them. The characteristics of the participants were as follows: average age 21.4 ± 1.50 years (range 21-26.5 years), height 179 ± 4.01 cm (range 174-189 cm), weight 81.7 ± 13.30 kg (range 65 - 100 kg), and body mass index 25.4 ± 4.40 kg/m^2 (19.4-30.3 kg/m^2). No athlete has a smoking habit. All participants followed their customary diets and performed their habitual professional and recreational activities. The time of experiment (10:00-12:00 am) was chosen to minimize the influence of circadian variations in salivary flow and composition. The test was performed at a minimum of 3 hours after breakfast and conducted in a well-ventilated room with an ambient temperature of 20-24°C, and relative humidity ranging between 40-55%. The subjects performed a Wingate anaerobic power test (WAPT) on Monark 814 E (Monark Exercise AB, Vansbro, Sweden). They warmed-up for 5 minutes at a pedaling rate of 60-70 RPM against a resistance equal to 20% of that was calculated for the subsequent WAPT. Two unloaded 5-second sprints were performed at the end of the 3rd and 5th minute of the warm-up period. The maximal pedaling rate (RPMmax) attained during the sprints was recorded. Following a 5 minute rest to eliminate any fatigue associated with the warm-up, they performed the WAPT against a resistance of 0.75 kg/kg body mass. They were instructed to pedal as fast as possible from the onset of the test. The resistance was applied when 70% of the previously recorded RPMmax was attained. They were verbally encouraged to maintain as high a pedaling rate as possible throughout the 30 second test duration. Heart rate was monitored throughout the warm-up, the test and the recovery method using a sport-

Table 1 - The values of plasma and salivary lactate (mmol/l).

Variables	Rest	R0	R5	R15	*p*
Blood lactate					0
Median	1.7	11.7	12.7	9.45	
Min-max	(1.22-2.53)	(1.22-2.53)	(8-16.20)	(5.20-13)	
Saliva lactate					
Median	0.21	0.24	0.27	0.43	0
Min-max	(0.9-0.30)	(0.11-0.49)	(0.9-0.56)	(0.22-0.63)	

Rest - before warm up, R0 - immediately after exercise, R5 - 5 minutes during recovery, R15 - 15 minutes during recovery.

Table 2 - Percentile of lactate increases compared with resting level.

Variables	R0 (%)	R5 (%)	R15 (%)
Blood lactate			
Rest	(600)	(650)	(455)
Saliva lactate			
Rest	(14)	(29)	(104)

Rest - before warm up, R0 - immediately after exercise, R5 - 5 minutes during recovery, R15 - 15 minutes during recovery.

Tester heart-rate monitor (Polar Electro, Kempele, Finland). The blood samples from all subjects were taken in tubes with sodium fluoride (NaF) from the antecubital region for lactate analysis, before the warm-up (rest), immediately after exercise (R0), 5 (R5), and 15 (R15) minutes following the WAPT in the inactive seated recovery period. Saliva samples were obtained at the same time with blood samples. Each subject was instructed to floss his teeth and thoroughly clean the oral cavity the night before saliva collection. Thirty minutes before the exercise test, each subject was given 500 ml of water to ensure adequate body hydration. The mouth was rinsed with deionized water immediately before saliva collection (resting or after exercise). In addition, each subject was instructed to empty his mouth of "old" saliva before spitting into a sterile container. Samples were kept at 4°C and transported to the laboratory. Once in the laboratory, samples were centrifuged and supernatants were separated and stored at –80°C. In order to reduce the variability to a minimum level and to avoid any possible interference, the clear transparent upper layer of the saliva samples was used for the lactate determination. Blood and saliva samples were analyzed using lactate PAP kit (Milchsaeure, Boehringer, Ingelheim, Germany) with a UV-Spectrophotometer (Philips Electronics, Eindhoven, The Netherlands) at the wavelength of 505 nm. Following performance of the homogeneity test; it was observed that the variances were not homogenous ($p>0.05$). While the Friedman Test was applied to compare the difference between Rest, R0, R5 and R15 measurements, Wilcoxon test was applied to determine from which groups the difference emerged. Significance level was accepted as $p<0.012$ by performing Bonferroni correction (a/k, k= comparison coefficient).

The differences were found significant between blood and saliva lactate values, at the measurements taken during rest and recovery (Friedman $p<0.001$). Blood lactate increased 10 mmol (600%), 11 mmol (650%), and 7.75 mmol (455%) at R0, R5 and R15, from resting value and decreased 3.25 mmol (26%) at R15, according to R5 value (Wilcoxon, $p<0.012$). Saliva lactate indicated an increase at R0 of 0.03 mmol (14%), R5 at 0.06 mmol (29%) and R15 at 0.22 (104%) mmol compared with resting level ($p<0.012$). Plasma and saliva lactate values are presented in **Table 1.**

According to Wilcoxon test, the differences between time points of measurement are significant $p<0.01$. There is no correlation between sample time values of blood and salivary lactate. The percentile of lactate increases with the sample time as presented in **Table 2**.

The study suggests that blood lactate peak occurs at the 5th minute of recovery, and saliva lactate peak occurs at 10-15th minutes of recovery.[1,3] Segura et al[2] and Chicharro et al,[4] found a good correlation between the concentrations of salivary and blood lactate, and they proposed the determination of lactate in saliva may be an alternative to the determination in blood at a maximum graded test on a cycle ergometer. Ohkuwa et al[1] cannot explain on physiological grounds why salivary lactate would significantly increase following a 400 m and 3000 m run, when compared with the basal level or whether salivary lactate concentration during recovery was significantly lower compared to blood lactate concentration, or whether the peak concentration of salivary lactate occurs later than the peak of blood lactate. One possible reason may be that salivary lactate is diffused passively through the salivary glands from the blood. At present, the origin of salivary lactate after exercise is not yet clear.[1]

In this study, during the post-exercise inactive recovery, plasma lactate showed an increase of 600% at R0, 650% at R5 and saliva lactate indicated

an increase of 14% at R0 and 29% at R5, plasma lactate was eliminated by 26% at R15, while saliva lactate increased by 104% at R15, compared to the resting values (**Table 2**). As can be seen, plasma and saliva lactate show different types of dynamics and there is not a linear relation between the 2. When the percentage increase of saliva lactate is compared with that of plasma lactate in our previous study as well as in present study, no relation between them is observed. However, following WAPT, saliva lactate levels at the post-exercise 15th minute in the previous and current studies were the same (0.43 mmol/l). Our results do not indicate the presence of any significant relationship between saliva and blood lactate values.[5]

It is expected that salivary lactate levels are parallel with plasma lactate levels with increasing workloads. Some studies suggest that determination of lactate in saliva may be an alternative to blood lactate investigation.[2] In both studies our plasma lactate and saliva lactate showed increases with supramaximal loading. There was no significant correlation between the peak lactate of saliva and blood increase, also percentile of lactate increases in blood and saliva, after supramaximal exercise.

In conclusion, it seems doubtful to estimate plasma lactate level by using saliva lactate level after supramaximal exercise.

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Target level controlled sedation. *An alternative to general anesthesia in endovascular treatment of intracranial aneurysms*

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Controlled sedation, commonly used to describe the process of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures by relieving anxiety, discomfort, or pain; while maintaining independent cardio-respiratory function is a term we refer to as target level controlled sedation (TLCS).[1] Endovascular therapy as interventional neuro-radiology (INR) is now an established therapeutic alternative to surgical clipping of some cerebral aneurysms.[2] In most institutions, an anesthesiologist is involved in the care of the patient during INR treatment.[3] The roles of the anesthesiologist in the INR suite are to monitor the patient, to provide appropriate anesthesia to facilitate the procedure, and to manage any complication that may arise. Embolization is usually performed under general anesthesia (GA) but in our study, we evaluate TLCS using 2 different agents in the anesthetic management of patients during the treatment of their cerebral aneurysm in the neuroradiology suite.

After approval of the Institutional Ethics Committee, and obtaining informed consent; 63 American Society of Anesthesiologists (ASA) I-III patients (37-68 years old, at least one week later than their subarachnoid hemorrhage) undergoing treatment for intracranial aneurysms by Guglielmi detachable coils (GDC) embolization were enrolled for our study. Patient demographic data, and current events prior to treatment, including the neurological status of the patient and duration of the procedure were documented. We established the intravenous access in the left hand. Patients were premedicated with midazolam 0.05 mg/kg and fentanyl one mg/kg intravenously (IV), and oxygen supplementation was stated at 2 L/min via nasal cannulae. After randomization, patients received an initial dose of propofol (10 mg/ml Fresenius-Kabi GmbH, Austria) 0.5 mg/kg (group P) or midazolam (one mg/ml, Dormicum, Roche, Basel, Switzerland) 0.05 mg/kg (group M), followed by an infusion of either propofol or midazolam as a sedative agent. The infusion rate was adjusted to maintain a sedation level of "5" by Ramsey Sedation Scale. If there was movement during the procedure, patients received