SPORTS MEDICINE AND REHABILITATION

Clinical and Experimental Studies
Review Articles

TARTU 1994
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QUALITY OF LIFE AFTER CORONARY BYPASS SURGERY

Aet Arak

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Abstract — The technical evolution of cardiac surgery in the last decades has enabled and in most cases enhanced survival also relieving incapacitating symptoms of patients with coronary heart disease. The improvement in the quality of this survival as subjectively felt by the patient has become most important to assess the justification of surgery. The article reviews current literature analyzing physiological and emotional state, intellectual function, performance of social roles and general satisfaction, i.e. quality of life (QL) after coronary bypass surgery.

Key words — bypass surgery, cardiac patients, quality of life

Introduction

QL has always played a role in the therapeutic objectives of medicine. While the evaluation of medical interventions is focused primarily on biological outcomes, judgements regarding therapeutic success also require consideration of the functional, psychological, and social burden associated with disease and its treatment (Staquet et al 1992). With increasingly large number of patients undergoing heart operations, both short- and long-term evaluation of functional status has become important, but relatively few studies have examined the patients’ own views of their functional status, employment, sexual functioning and emotional state (Pinna Pintor et al 1992), although it has been showed that evaluation of patients’ may assist in planning interventions and favourably enchanche their therapeutic outcome (Gilutz et al 1991). Consideration of QL in the medical care context is related to the ways in which a patients life is affected both by illness and its care (Wenger et al 1984, Wenger et al 1989). In contemporary cardiology, a major contributor to the emphasis on quality of life outcomes is the increased prevalence of chronic cardiovascular...
diseases. QL is of particular importance in any chronic illness, in that the therapeutic goals are not a cure, but rather an alleviation of symptoms, an improvement of functional capacities, limitation of the progression of the disease, and a lessening of the adverse psychologic consequences that may lead to unwarranted invalidism (Wenger et al 1990).

Quality of life: definition and measurement

Although there is no generally agreed upon definition, much progress has been in recent research in defining QL. In 1976 the term "quality of life" received a listing in the medical literature in \textit{Index Medicus} (Prevost et al 1993). Health status, functional status and quality of life are three concepts often used to refer to the same domain of "health" (Patrick et al 1990). Ferrans and Powers defined quality of life as a person's sense of well-being that stems from satisfaction or dissatisfaction with the areas of life that are important to him/her. It has been emphasized that for a meaningful description of QL, the objective observable change must be related to the subjective meaning given by an individual to the change (Najman et al 1981). Despite the difficulty in measuring the wide range of a concept by which one could mean QL, several studies could conclude that components constructing the quality of patients' life are (1) Physiological state (2) Intellectual function (3) Emotional status (4) Performance of social roles (5) General satisfaction (Walter et al 1992). The major reasons why clinicians, health administrators and patients are keenly interested in the effects of medical interventions on QL include the following:

1) As chronic disease have become increasingly prominent the goals of health care have changed. Beside the traditional outcome indicators of mortality and morbidity the data reflecting patients' self-perception of their health status are necessary to make decisions about the outcomes of care provided to those with chronic disabling illness (Jette 1993).

2) The population in developed countries is aging. Elderly persons with chronic disease represent an increasing percentage of all persons served by health care professionals (Selker et al 1988) and coronary disease is the cause of death of two-thirds of them (Meshan et al 1992).

3) Cost containment has become a dominant theme in health-care worldwide. It has become an economic imperative to critically
examine the relative costs and benefits of different health interventions (Jette 1993).

Packa (1989) asserted that attention should be given to considering subjective, objective and disease-specific indicators in health-related studies when measuring quality of life. Measures of health status should be designed to evaluate symptoms, functional capacity, perceived illness or well-being, patient expectations, emotional function, requirement for medical or other services, and social requirements. The indicators must be quantifiable and reliable (Wenger et al 1984). There is no standard approach to measurement of these variables and it has been constantly under severe discussions. Several reliable and valid instruments are available for almost any QL component that needs to be measured (Stewart 1992). Some of them that have been used in measuring aspects in cardiac surgery patients include the Nottingham Health Profile (Caine et al 1991), The Profile of Mood States (Jenkins et al 1983), The Psychological adjustment to Illness Scale (Raft et al 1985), the McMaster Health Index Questionnaire (Packa 1989) and the Ferrans and Powers Quality of Life Index (Ferrans et al 1992) (Table.1).

Table 1. Examples of Tools Used in Measuring QL after cardiac surgery

<table>
<thead>
<tr>
<th>Tool</th>
<th>Length (Items)</th>
<th>Administration</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nottingham Health Profile</td>
<td>45</td>
<td>Self-report</td>
<td>Measures 6 domains of experience: pain, physical mobility, sleep, emotional, energy, and social isolation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Measures 7 domains of daily life: employment, household work, personal life, hobbies, relationships, vacations, and sex</td>
</tr>
<tr>
<td>Profile of Mood States</td>
<td>65</td>
<td>Self-report or interview</td>
<td>Measures 6 dimensions of affect or mood: tension-anxiety, anger-hostility, depression-dejection, vigor-activity, fatigue-inertia, and confusion-bewilderment</td>
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<td>------------------------</td>
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<tr>
<td>Psychosocial Adjustment to illness Scale</td>
<td>46</td>
<td>Self-report or interview</td>
<td>Measures 7 life domains: health, domestic atmosphere, family relations, social environment, sex, work, and psychologic distress</td>
</tr>
<tr>
<td>McMaster Health Index</td>
<td>59</td>
<td>Interview</td>
<td>Measures 3 dimensions of health: physical, social, and emotional. Designed to measure functioning rather than capacity for functioning</td>
</tr>
</tbody>
</table>
Ferrans and Powers
Quality of Life Index

Self-report or interview

Measures 4 major domains: health and functioning, family, socioeconomic, and psychological/spiritual Measures satisfaction with various domains of life and the importance of each domain

From Prevost et al. 1993.

Parameters obtained from the questionnaires need to be correlated with physiologic outcomes and morbidity and mortality. More information is necessary concerning methodologic aspects with regard to the design and the administration of the questionnaires. The strengths and weaknesses of the different modes of administration are summarised in Table 2.

Table 2. Modes of Administration of QL Measures

<table>
<thead>
<tr>
<th>Mode of Administration</th>
<th>Strengths</th>
<th>Weaknesses</th>
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<tbody>
<tr>
<td>Interviewer</td>
<td>Maximizes response rate. Few, if any missing items. Minimizes error of misunderstanding</td>
<td>Requires many resources, training of interviewers. May reduce willingness to acknowledge problems. Limits format of instrument</td>
</tr>
<tr>
<td>Telephone</td>
<td>Few, if any, missing items. Minimizes errors of misunderstanding. Less resource intensive than interviewer-administered mode</td>
<td>Not available everywhere</td>
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<tr>
<td>----------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Self</td>
<td>Minimal resources required</td>
<td>Greater likelihood of low-response rate, missing items, misunderstanding</td>
</tr>
<tr>
<td>* Surrogate responders</td>
<td>Reduces stress for target group (very elderly or sick)</td>
<td>Perceptions of surrogate may differ from target group</td>
</tr>
</tbody>
</table>

*A close relative who responds on behalf of the patient who is not capable of answering*

*From Guyatt et al. 1993.*

### The Outcomes of Coronary Artery Bypass Surgery in Quality of Life Context

Until recently the goal of coronary artery bypass surgery (CABS) was to enable or enhance survival, and sometimes to relieve incapacitating symptoms. Technical advances of the last decades made it routinely possible to meet these aims and prolong the survival (Walter et al. 1992). Still, to survive does not always mean happy and meaningful life. Further the main components constructing QL after CABS is reviewed.

#### (1) Physiological state

Regarding death and ischaemic events after CABS approximately 77% patients are free from all ischaemic events at 5 years and 50% at 10 years (Kirklin et al. 1989). Over 90% survive 5 years and approximately 80% survive 10 years (Sergeant et al. 1990). In one of the institutions freedom from angina at 1, 5 and 10 years respectively was 98%, 89%, 69% and 38% if internal mammary artery (IMA) graft was used. Freedom from sudden death was 99.8%, 99% and 97% at 1, 5 and 10 years (Sergeant et al. 1991). 15 year survival was
around 63% at 15 years when the IMA was used in another study (Johnson et al 1989). Approximately 10% of vessel occlusions when saphenous veins as grafts are used within the first weeks after surgery and are caused by technical problems or thrombosis at the distal graft anastomosis (Kirklin et al 1991). Angiographic studies have shown that a 2% per year vein-graft attrition rate from the first to the seventh postoperative year increases to approximately 5% per year from the seventh to the twelfth year due to intimal hyperplasia within saphenous vein graft (Loop et al 1986). IMA grafts have been shown in recent studies to have superior early and late graft patency: between 85% and 95% of IMA grafts are patent 7 to 10 years after surgery (Loop et al 1986). About 2.5 to 10% patients may need a reoperation in 5 years (Kirklin et al 1986), but about 66% need it in 15 years (Kirklin et al 1989). In the United States, reoperation for coronary artery disease represents about 7% to 8% of the coronary surgery performed annually (Gallotti et al 1991). Operative mortality was found to range from 0.4% to 2.2% (Gersh et al 1988); for reoperations it was 4% (Loop et al 1981, Kirklin et al 1986). Evidence from all randomized trials has shown the distinct benefit of surgical over medical treatment in long term survival and relief of ischaemia in patients with left main coronary artery stenosis, multivessel disease, left ventricular dysfunction and severe symptoms (CASS 1983, Gersh et al 1988). At 7 years of follow-up the CASS group demonstrated an 88% survival rate in the GABS group, compared with a 65% survival rate in medically treated group; an increase of 20% to 50% in maximal exercise performance also was reported in the surgery group (CASS 1983). However, at 10 years of follow-up, there was little, if any difference in freedom from angina and activity limitation and in use of antianginal medication between the two groups (Gersh 1992). The completeness of revascularization has been found to be an important predictor of long-term survival and functional status after coronary bypass surgery (O’Keefe et al 1993). Functional benefit is somewhat less in women. Surgical mortality is increased in women, and the long-term effects upon QL and recurrence of symptoms appear less impressive than in men (Gersh et al 1983). In the study by Loop et al women had twice the mortality of men, with the greatest difference seen in age-matched group 60 years or younger (Loop et al 1983). This study showed that body surface area, regardless of gender, was the strongest predictor of operative risk, suggesting that the historically described less favourable outcome in women may have been caused by generally smaller body surface area and smaller caliber of coronary arteries (Loop et al 1983). Several recent studies have
suggested that differences in outcome may not be necessary due to technical difficulties but may be caused by referral bias: women are referred to surgery at more advanced stage of coronary disease (Tobien et al 1987) and they tend to be older than male patients (Uhan et al 1990). Sicker (severe cardiac and noncardiac diseases) and elderly patients undergo surgery, furthermore, GABS is justified in the very elderly as the health-related quality of extended survival is at least as good as that of younger patients (Walter et al 1994). Results of Albes et al demonstrate the significant difference in early operative mortality between elderly and younger patient groups (75 years: 6.9%; <75 years: 2.3%) (Albes et al 1991); similar results have been shown in another study with corresponding values 7.2% versus 1.45% (Mohan et al 1992). The clinical situation among survivors in the older group has improved considerably: over 75% of the patients are free from angina (NYHA I) or can exercise properly (NYHA II) (Albes et al 1991).

(2) Intellectual function.

The main contenders as the mechanism causing the neurological and intellectual (neuropsychological) deficits in patients undergoing CABS are microemboli (particulate and/or air) and/or perfusion related damage as a result of extracorporeal circulation (Newman 1992). Microembolic events are particularly in evidence at the time of cannulation and at the inception of bypass (Pugsley 1989, Pugsley et al 1989). According to Newman 24% of patients show neuropsychological deficits 12 months after surgery and that these deficits are related to age and the duration of bypass (Newman 1992). A history of diabetes, evidence of mural thrombus, positive oculopneumoplethysmography findings, aortic calcification, recent myocardial infarction and postoperative arrhythmias all correlate with increased risk of permanent neurologic deficit for the patient undergoing coronary bypass (Lynn et al 1992, Tuman et al 1992). The prevalence of cerebrovascular accidents in the patients having undergone CABS in United States has been documented to be from 2% to 16% (Breur et al 1981, Coffey et al 1982, Craver et al 1982). The reported incidence of stroke ranges from approximately 1% to 5% (Loop et al 1988, Kuroda et al 1992), but among patients ≥75 years the incidence of stroke or transient ischemic attack was 8.6% (Albes et al 1991). Neurological abnormalities in another study were seen in 79%
of 298 CABG patients in the early postoperative period; minor neuropsychological impairment may persist at 6 months, but only 27% of patients are symptomatic and only 2% are seriously disabled by intellectual dysfunction (Shaw 1992). Ellis and associates found that 75% of 30 CABS patients had deterioration in some aspect of intellectual function 7 days after surgery. At 4 weeks 17% were still impaired but all had returned to the normal by the end of 6 months (Ellis et al 1980). The aspects of cognitive function observed most commonly to deteriorate after CABS include short-term memory, new learning ability, attention span and psychomotor speed (Shaw 1992). Concerning long-term prognosis for CABS patients with postoperative intellectual impairment, detailed studies have been few. Ellis et al (1980) considered, that the lack of long-term outcome dysfunction indicated that low flow-rates and low arterial pressures could safely be used during CABS. Some authors have found, that much of the early deterioration resolved within 6 months (Ellis et al 1980, Savegeau et al 1982). 20% of patients with early neuropsychological deterioration after cardiac surgery were still impaired at 6 months (Savegeau et al 1982). The delayed deterioration can occur following a cerebral hypoxic-ischemic insult, but it cannot automatically attributed to cardiac surgery (Shaw 1992). Patients with postoperative neurologic events are reported to have ninefold increase in mortality - 35.7% versus 4.0% (Tuman et al 1992).

(3) Emotional state

Some patients fail to adapt to the CABS and do not function well psychologically. It has been shown, that emotional impairments greatly outnumber physical impairments: a high level of pre-operative state anxiety and a post-operative higher status of anxiety could be considered as possible predictors of functional cardiac complications (Pinna Pintor et al 1992). In some studies has been demonstrated, that despite improvements in physical symptoms, poor adjustment and persisting of worsening psychological complaints have been observed in 25 - 40% of post-surgical patients (Frank et al 1972, Heller et al 1974, Zyzananski et al 1981, Horgan et al 1984). Another study in a sample of 340 patients requiring CABS showed, that the level of usual daily activity significantly increased over a 6 months period and among the many factors associated with higher levels of daily activity were higher sense of well-being, lower depression and anxiety (Stanton et al
Anxiety and depression have been reported to be closely associated with decreased quality of life (Hirsch et al 1991). Anxiety is the main symptom before and immediately after surgery (Langosh et al 1992), but continuing postoperative fatigue, atypical chest pain and disorders of sleep and libido etc change the preoperative anxiety about surviving to postoperative depression (Gundle et al 1980). Boudrez et al have demonstrated that 10-20% of patients exhibit dysfunctional psychological states postoperatively (Boudrez et al 1992). The symptoms having highest loadings in the postoperative depressive syndrome are lack of energy, feelings worry, pessimistic ideas, blaming their physical condition for being nervous and narrowed thinking (Langosh et al 1992). The strongest predictor of emotional distress after operation is pre-existing anxiety or depression (Kos Munson et al 1974), though patients themselves relate emotional distress to their lack of knowledge of what to expect of surgery. About one third of 249 patients felt that their knew enough going back to work, resuming sexual activity and the possible physical symptoms that might be expected (Stanton et al 1984). Magni et al demonstrated that about 25% of patients were found to have persisted or new psychological distress (high scores especially on depression and anxiety scales) one year after surgery (Magni et al 1987). According to the results of Perski et al 1 year after the operation the medical effects (angina and dyspnea relief) were excellent, but those results were not coupled in general health status and subjective well-being: 50% of the patients complained of depression, nervousness and irritability (Perski et al 1991). 3 years after CABS surgery only 5% of patients were depressed in a group with psychologic intervention in contrast to 30% in those without such intervention (Walter et al 1991). In an analogous study was found, that depression was alleviated in the rehabilitation group (Engblom et al 1992), but it has been stated, that the response to rehabilitation, in turn, depends on the emotional state of the patient (Cay 1989). Sexual activity decreased from 67% once a week to 38% once a week at 9 months and further deterioration at 3,5 years (Kornfeld et al 1982). After preoperative decline sexual interest increased in 85 % and sexual activity in 49% among CABS patients one year after the operation (Langelduccke et al 1989). Contradictory are data from Finnish study: 12 months after surgery only 9% of rehabilitation group and 10% of hospital-based treatment group had experienced an improvement in sexual life, a worsening was experienced correspondingly by 42% and 51%(Engblom et al 1992). The last study also demonstrated that subjects who felt that coronary heart disease (CHD) had decreased their
sexual activity before surgery were less satisfied with the postoperative sexual life and experienced more often a worsening of their sexual life than patients in whom CHD had not impaired sexual function (Engblom et al 1992).

(4) Performance of social roles and return to work

Employment status after CABS has been of interest as going to work is a vital socioeconomic function and its non-continuation after operation can lead to diminished self, familial and social esteem and financial hardship. It reflects not only the absence of symptoms with satisfactory exercise capacity but also the emotional stability required to get up, meet people, create and earn for the happiness of the patient and his dependents (Walter et al 1992). In a review of the literature by Russell et al the rate of return to work after CABS varies between 40% and 90% (Russell et al 1986). Different national levels of employment and wide social and insurance benefits makes it difficult to compare these studies (Ägren et al 1993). 71% of the men < 65 of age were working one year after CABS compared to 23% 1 month before surgery (Ägren et al 1993). Another report demonstrated a 60% rate of resumption of work after CABS (Frick et al 1979). Comparison between all retired and all working patients demonstrated that 63% of working individuals were discharged within 8 days of operation, in contrast to only 44% of retired persons (Finkelmeier et al 1993). Those who do not have the external pressure of a job commitment and provision of financial support may not experience the same compulsion to return to full activities as quickly (Finkelmeier et al 1993). A clear positive correlation existed between the expected desire to work and actual return to work: of patients wanting to work, 79% actually worked, whereas 97% of those who did not want to work retired (La Mentola et al 1979). Return to work was 80% in those working until the operation as compared with 44% who had stopped working earlier (Walter et al 1992). For self-employed patients and white-collar workers return to work was 73% and 69% respectively corresponding to only 50% for blue-collar workers (Walter et al 1992). Occupational problems are greater among blue-collar workers, less-educated patients and those with more serious medical complications (Rankin 1992). Despite adequate exercise capacity and better relief of angina through
surgical than medical treatment, in three studies employment was comparable if not better after medical treatment (Hammermeister et al 1979, CASS 1983, Russell et al 1984). But is employment after CABS a measure of the quality of life (Walter et al 1992)? Evidently, depending on disability and retirement politics, return to work varies from country to country (Walter 1988). A preference may be given instead of working to retire early in life with disability benefits and to enjoy pleasant time at home and outdoors with the family and friends, a possibility provided by the improvement in physical capacity and in the newly gained independence due to the operation (Prevost et al 1993). Such patients find their quality of life satisfactory and the surgical outcome is considered beneficial regardless of the employment status (David 1978). Psychosocial factors tend to be more predictive than do physiologic factors. Predictors that were identified repeatedly included preoperative employment status, type of occupation, educational status, nonwork income, and preoperative perceptions (i.e. patients who do not expect to be able to return to work after surgery usually do not return even if they have no physical obstacles (Prevost et al 1993). In contrast to return to work, family relationships and social activities have constantly been shown to improve after CABS (Mayou et al 1987, Langduccke 1989). Dependence decreased in 73% patients and for interest and capacity in household work also improved substantially for housewives (Ross et al 1978). Marital satisfaction was mostly unchanged (Jenkins et al 1988) and intimacy with children increased in about half of the patients (Mayou et al 1987).

(5) General satisfaction

General satisfaction is markedly influenced by patients family function. Spouses seem to have the ability to influence patient recovery both positively and negatively (Artinian 1993). 88% with CABS patients living with spouse were "very happy" or "happy" 1 year after operation (Jenkins et al 1988) and pleasure in life had improved for 77% of them when asked 3,5 years after the operation (Kornfeld et al 1982). O'Connor (1983) reported that cardiac surgery perceptions of patients' health were negatively influenced by their spouses' fear of injury during rehabilitation. It has been demonstrated that increased support from partner after cardiovascular event was associated with decreased stress for patients and partner support was the strongest correlate of relational well-being for patients (Yates 1989). Patients
reported better mood and sense of control within half a year after surgery, it took more than a year until they felt more active and sociable (Ägren et al 1993). Many of the patients had developed new standards of reference in life as suggested by the responses of more than 1/4 of the patients that they became calmer and more tolerant and they got more joy out of life (Ägren et al 1993).

Several studies have stressed the favourable effect of comprehensive cardiac care (CCC) on QL after CABS. It is based on physiological, psychological and social aspects and includes a close clinical follow-up of patients (Kellermann 1992). Risk factors are eventually modified within the program: these includes a complete cessation of smoking, dietary measures and the control of hyperlipidemia, hypertension and diabetes (Gersh 1992).

Conclusion

Although the final result of CABS is considered to be a lessening of the morbidity and mortality of treated patients, but it must also encompass efforts to help patients lead more meaningful and better lives. Consideration of quality of life outcomes may help in medical judgements of what is best for a specific patient in a specific situation, may help patients in their decisions for choice among therapeutic interventions and contribute to reasonable decisions in using health care resources. In spite to intensive research in developed countries concerning QL after cardiac surgery the similar data in Estonia are almost missing, although CABS has been performed since 1974. For improving the efficacy of surgeons', cardiologists' and general practitioners' work the corresponding information would be extremely helpful.

Acknowledgements

The article has been prepared for issuing during my stay at the Dept. of Cardiology of Karolinska Hospital, Stockholm, as a scholarship holder from the Swedish Institute.

My sincerest gratitude to Prof. L. Rydén, Head Dept. Cardiology of Karolinska Hospital for his kind assistance and stimulating support in everything; to Prof. P. J. Walter and R. Mohan from the University Clinic of Antwerp, and Prof. J. Maaroos, my tutor,
for having patiently introduced me with the topic of quality of life after cardiac surgery.

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Kokkuvõte

Aortokoronaarne sunteerimine südame isheemiatõve kirurgilise ravina on viimastel aastakümnetel tänu järjest täiuslikematele tehnilistele võimalustele jõudsalt arenenud ning võimaldanud operatsiooni läbiteinud haigetele pikaajalise, sageli vähese või talutavate komplektsionidega elulemuse. Operatsioonijärgne elu kvaliteet kui tehniliselt kompleksne ja väga kuluka protseduuri olulisim tulemus patsiendi poolt hinnatuna on nüüdseks kogu maailmas tõusnud tähelepanu ja teaduslike uurigute keskpunkti. Artikkel annab ülevaate aortokoronaarse sunteerimise läbipealine patsientide lõikusjärgset elu kvaliteeti käsitlevast kirjandusest, kusjuures peatutakse füsioloogilisel, emotionsaalset ja intellektuaalset seisundil, sotsiaalsele rollide täitmisel ning üldisel rahulolul.
ELECTROTHERAPY: A PART OF REHABILITATION OF HAND AND LEG FRACTURED PATIENTS

Maire Audova

Department of Sport Medicine and Rehabilitation, University of Tartu, The University of Tartu Hospital

Abstract — Several types of electotherapy have been used in the rehabilitation of patients after fractures of hand and leg for many years. The advancement of medical technology has given some new physical factors like interference currents and low frequency magnetic fields therapy which have been proved effective in the management of pain, oedema and diminished blood flow.

Key words — alternating currents, direct currents, high frequency, low frequency, magnetic field, physiotherapy

Introduction

In the department of physiotherapy of Maarjamõisa Hospital electrotherapy has been used for many years in order to speed up healing of hand and leg fractures. In the 1970s we began electrotherapy only after the removal of the plaster cast, and we usually did not apply the current to the fracture site itself but to the secondary site of the injury or to the cervical or lumbosacral vegetative ganglions with the view to increase the blood supply in the extremities. Direct applications were painful and would increase oedema. Sudeck's dystrophy - diffuse osteonecrosis, swelling, pain, muscular atrophy and functional impairment - was not a rare complication. Its management was laborious and time consuming. In 1980 our department got a new apparatus - a Russian "Poljus" which produces low frequency magnetic fields, and in 1982 a Polish "Interdyn", which produces interference currents. Both methods are now irreplaceable in the treatment of fractures - we can apply the physical factor directly to the injured limb, it is easily tolerated by the patient and has been very effective.
Therapeutic effects of the physical factors used

Magnetic fields (MFs) used in electrotherapy are either static magnetic (SMFs), low frequency (LFMFs) or high frequency fields (HFMFs). The advantages of MFs include non-invasiveness and noncontact (Weil 1988, Pienkowski et al. 1994). MFs stimulate microcirculation and transcapillary metabolism which in turn facilitate regenerative processes, particularly in the bones (Ulaskchik 1986). MFs penetrate all the tissues, including the bones, by this way elevating the concentration of calcium ions in the chondrocytes and simultaneously increasing the blood flow (Chvojka 1985). A stimulative effect to the healing of fractures has been found both in the case of SMFs and LFMFs. Nikolski et al. (1987) reported that SMFs have an anti-inflammatory, analgetic and oedema-reducing effect, and that they also speed up the regeneration of the bone tissue. Demetskaya (1987) achieved a rapid decrease in oedema and pain when using SMFs. Boltrukevitch et al. (1980) observed that after 5-6 SMFs sessions pain and oedema diminished, blood flow increased and the healing of the fracture progressed faster. Tkatchenko et al. (1980) noticed that the use of SMFs contributed to the formation of normal structured bone tissue. Fedorova et al. (1980) report an acceleration in the formation of the callus and a decreased oedema. More effective than SMFs are LFMFs. Dormidontov (1980) found that oedema became faster resorbed when LFMFs were used in the treatment. Freedman (1985) used pulsed electromagnetic fields in the treatment of delayed and non-union fractures and suggested that the frequencies should be changed if the bone healing was insufficient. It is important that LFMFs suit also for the patients with metal devices or metal foreign bodies (Singer et al. 1977, Saveliev et al. 1980, Ulashtchik 1986). Yasnogorodski (1987) is of the opinion that LFMFs are rather a week factor and recommends its use in the cases where other methods are contraindicated. Speranski (1975) has described the sedative effect of MFs to the central nervous system: the sleep will become sounder and the emotional stress will decrease, and that is important in order to avoid Sudeck's dystrophy (Mittelbach 1987).

The commonly and widely used electrotherapeutic factors are low-tension and low-frequency direct and alternating currents. All these methods: diadynamic currents (DDC), interference currents (IFC), amplipuls-therapy (sinusoidal modulated currents SMC), Träbert's currents (TC) and transcutaneous electric nerve stimulation (TENS), are well-known in the treatment of pain. Zuckerman (1990)
considers that the effect of low-frequency currents may possibly be related with the transmission of these electrical impulses, and that it so-to-say blocks the pain impulses from passing through the gate (Melzac et al. 1967). Uhlemann et al. (1989) thought that the goal of physiotherapy in the treatment of osteoporosis is the depression of pain, activation of muscles and stimulation of the metabolism of the bone tissue. They recommended DDC, ultrasound (US) and impulse currents. In the treatment of retarded recovery patients Dorian et al. (1989) used IFC as a factor for acceleration of osteogenesis. As said above, the method is non-invasive. Nikolova (1991) preferred IFC to other factors like US, DDC, SMC, LFMFs, electrophoresis. Useful effects of IFC include normalization of the blood and lymph flow, reduction of oedema and regulation of osteosynthesis, as pointed out by Ulashtchik (1986). Yasnogorodsky (1987), too, pointed to the analgetic effect of IFC in the treatment of posttraumatic pain, but he believed it to be more effective if IFC is used in the acute stage after the injury.

Using of other methods of impulse currents is a little complicated. In the acute stage after the fracture, during bone immobilization, the placement of electrodes is made more difficult by plaster cast, and special windows are needed, but even worse is the fact that electrodes when placed on the spot can increase pain.

**Conclusions**

A better consolidation of bone fractures, achievement of an analgetic effect, reduction of oedema, prevention of stiffness of joints and muscular atrophy - these are purposes of electrotherapy. Since after fracture the injured region is fixed with an immobilizing plaster cast, and the limb is swollen and painful, it is difficult to use methods that require placement of electrodes to the injured area. Today we have two alternative methods, magnetic fields and interference currents, both of which are non-invasive and noncontact but convey the physical factor directly to the site of the injury. The effects of LFMFs and IFC reported in literature were confirmed by our experience in the fracture-repair process.

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Kokkuvõte

Käe- ja jalaluude murdude taastusravis on paranemise kiirendamiseks kasutatud elektriravi juba aastaid. Meditsiini tehnoloogia areng on andnud uusi tõhusaid faktoreid - madalsageduslik magnetväli ja interferentsvoolud - mis on osutunud efektiivseks murrujärgse turse ja valu vähendamiseks ning häiritud verevarustuse parandamiseks.
FIRST EXPERIENCE OF ARTHROSCOPY: SUCCESS, DIFFICULTIES, COMPLICATIONS

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The purpose of this paper is to estimate our first experience and to express our opinion for improvement of our results.

152 arthroscopies of knee joint were made with several indications as: meniscal tears, unclear synovitis, cartilage damages, loose bodies, Hoffa's disease, ACL and PCL ruptures, plica syndrome etc. Different portals were chosen by different surgeons: central, lateral, medial. On 25 cases surgeons were forced to finish the operation through miniarthrotomy. The purposes were:
- too big loose body to extract through the usual portal for arthroscope
- difficulties with ACL or PCL suture
- lateral meniscus tears in a very tight knee

Success.

1. Arthroscopically established diagnoses are more believable.
2. Postoperative use of nonsteroid antiinflammatory drugs (NSAID) are enough for painless care.
3. Recovery after arthroscopic procedures are more fast than after open procedures.

Difficulties.

1. Poor choice or absence of some instruments or equipment.
   - liquid supply system: we are forced to use liquid from 0.5L glass bottles and are in technical difficulties with intraarticular pressure.
   - shaver
   - tourniquet system: we use rubber tourniquet on thigh.
   - leg holder
   - videoprinter
2. Minor mistakes due to incorrect choice of portal. It may lead to poor visibility, to difficulties to handle instruments.
Complications.

1. Phlebalgia or phlebitis in 5 cases, which were due to (in our opinion) rubber tourniquet. Fortunately they all were treated conservatively in few days.
2. Compartment syndrome in 1 case, which did not need any treatment.
3. Adhesive scar of portal 1 case, treated by electrophoresis with lidase.
4. Infected joint on 2 cases

Conclusion.

The use of arthroscopy permits: to establish exact diagnosis, fast and easy recovery for all kind of activities. A further improvement of skill, knowledge and equipment is needed to gain better results.
ON THE PROPER CUFF PRESSURE FOR VENOUS OCCLUSION

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Abstract — Eleven surgery patients were tested in order to examine cuff pressure value needed for the venous occlusion. Measurements were taken from the upper arm as well as the thigh. The proper value of the cuff pressure for venous occlusion would lie between 55 - 60 mmHg.

Key words — cuff pressure, plethysmography, venous occlusion

Introduction

At venous occlusion plethysmography and venous occlusion impedance plethysmography methods (Anderson 1984) a cuff inflating pressure is needed that would close (occlude) veins under the cuff remaining arteries opened.

The aim of the present work was to study whether it is possible to find common cuff pressure value for most of the people, satisfying the described condition.

Experiment

Investigations were carried out on a mixed contingent of healthy people and vascular surgery patients. Eleven persons of ages from 14 to 61 years were studied. Several cycles of measurement were done with each patient, separately on legs and on arms. The pressure cuff was placed around the upper arm or the thigh.

The pressure in cuff was raised so high, that the occlusion of veins was quaranteed. Decreasing the pressure in the cuff, a pressure value was dermined at which the veins opened. The arterial pressure (using the common Korotkoff method) was estimated, too. The flow of the blood in veins was watched at a distal from cuff position by a Russian made Doppler-echo blood velocity meter ISKN. The received
data were collected by a collector of biomedical data based on personal computer (Jaksman et al. 1992 a, b). An example of one experiment is given on fig. 1. Here curve a represents cuff pressure, black-covered areas b and a horizontal line at bottom of the figure represent the output of the Doppler-echo velocity meter. Point A is a starting points of the experiment - cuff is inflated up to starting pressure, at point B the cuff pressure has value at which the blood flow in veins begins. Thus at the beginning of flow in veins the corresponding pressure in the cuff was easily estimated using computer analysis of the collected data.

Figure 1. Representation of the computer screen from an experiment for estimation of the cuff pressure for venous occlusion. Curve a - cuff pressure; Black area b - Doppler-echo meter indicates non-zero velocity of the venous blood flow; at point A cuff is inflated up to starting pressure; at point B the cuff pressure has the value at which the flow in vein begins.

Results

The results of the investigations are given on fig. 2, where the adjacent columns represent correspondingly: the maximal point B cuff pressure for arm, the maximal point B cuff pressure for leg and the (separately measured) diastolic arterial pressure of the same patient. From the all point B cuff pressure values, that were estimated in
different measurement cycles for a patient, only the maximal values are depicted on the figure 2.

![Diagram of veins occluding pressures and arterial pressures for 11 persons. Explanation see in text.](image)

**Figure 2.** Diagram of veins occluding pressures and arterial pressures for 11 persons. Explanation see in text.

**Discussion and conclusion**

Though Anderson (Anderson 1984) has suggested that the common for most people cuff pressure for occlusion exists, he did not add characteristics of dispersion of the necessary pressure. Therefore we decided to check Anderson's suggestion.

It seems to be evident see fig.1, that at the cuff pressure exceeding point B pressure, the veins under the cuff are fully collapsed (i.e., occluded). By the other side, the arteries do not collapse until the cuff pressure is less that the diastolic arterial pressure (possibly they can be deformed a bit by the cuff pressure). Figure 2 shows that despite the dispersion of arterial pressures and veins occluding pressures on different people, in our experiments a "corridor", between the maximal point B pressure and diastolic arterial pressure can be estimated. The proper value of cuff pressure for venous occlusion would lie in this "corridor", i.e. between 55 - 60 mmHg. This is the pressure value that would be common for most of the people (even with persons having...
vascular diseases), that fully closes the veins and has minimal influence on arteries.

References


Kokkuvõte

Antud töö eesmärgiks oli leida veenide rõhumansetiga tekitatava oklusiooni jaoks (mõõtmised tehti mõlemal käel ja jalal) mansetirõhu väärtuste piirkond, mis tagaks inimese veenide sulgemise arterite avatuks jäämise korral. Katsealuste grupis oli 11 inimest vanuses 14 - 61 eluaastat. Meie katsed kinnitavad, et selliseks mansetirõhu piirkonnaks on 55 - 62 mm Hg.
KNEE ANTAGONIST MUSCLE COACTIVITY DURING FATIGUING EXERCISE

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Abstract — Knee antagonist muscle coactivation was studied in 14 ice hockey and 14 football players during 1-minute fatiguing isokinetic exercise at joint velocity of 250 deg/s. Surface electromyograms (EMG) were recorded from the vastus lateralis, vastus medialis, rectus femoris and biceps femoris muscles of the right leg. Coactivity was calculated as a ratio of average electromyographic activity (μV) during the antagonist phase of the work of muscle to the average electromyographic activity of the muscle as agonist for each joint angle studied. Fatigue was measured as a shift in median frequency (Hz) of the myoelectric power density spectrum to the lower frequencies which was calculated with the aid of the Fast Fourier Transform. In football players (high hamstring/quadriceps peak torque ratio) increased biceps femoris coactivation was observed at the terminal 30 deg of knee extension (p < 0.01) and low rectus femoris coactivity throughout the full range of movement (p < 0.05) as compared to ice hockey players (low hamstrings/quadriceps peak torque ratio). Muscular fatigue led to a decrease in antagonist muscle coactivation at initial and terminal phases of joint movement (p < 0.05). It was concluded that the constant opposing torque strategy in antagonist muscles recruitment gets lost when muscle become fatigued as well as active dynamic break of knee movement. Training of the hamstring muscles is recommended for the ice hockey players.

Key words — antagonist coactivation, hamstrings, quadriceps, muscle fatigue
Introduction

Interest to the coactivation phenomenon has continuously increased during last decade, because antagonist muscle coactivation serves an important role in maintaining joint stability by generating opposing force (Hsieh et al. 1976, Schoemaker et al. 1982, Hagood et al. 1990). The coactivation of the antagonist may vary from 5% up to 40% of its maximal activity when acting as agonist (Solomonow 1990). It indicates that the opposing force generated by the antagonist muscle is not negligible. The ability of a muscle to co-activate depends on its force-generating and viscoelastic properties as well as movement amplitude and velocity (Madsen et al. 1983, Baratta et al. 1988, Hagood et al. 1990). A direct link between agonist-antagonist set of muscles considered in many studies (e.g. DeLuca et al. 1982a, 1982b, 1987) suggests that the activity level of the agonist models the response of the antagonist.

No studies have been performed to examine coactivity phenomenon in fatigued muscles. Force generation capacity of a muscle decreases when it becomes fatigued (Hagberg and Ericson 1982, Moritani and Muro 1987). The objective of this study has, therefore, been to clarify the effect of the muscular fatigue on antagonist muscle coactivation. Force imbalance over a given joint may increase the risk of joint/ligament injury and such a knowledge may be of importance.

Subjects and Methods

Subjects
Twenty eight young male subjects (football and ice hockey players; age 22.84 ± 2.06 years, height 174.52 ± 4.89 cm, weight 73.01 ± 3.42 kg; mean ± SE) without any known knee pathology were involved on their informed consent in this study. The physiological and biomechanical aspects of the research were explained to the subjects before participation. Average hamstrings/quadriceps peak torque ratio in ice hockey players (group A, n=14) was remarkably lower than in football players (group B, n=14), 44.47 ± 3.69% vs. 61.05 ± 4.03% (p < 0.001), respectively.

Instrumentation
The subject was seated, leaning against a backrest inclined at 20° from vertical and the seat inclined 20° from horizontal so that angle
in hip joint was 90°. Only the right leg was tested. The axis of the knee was aligned with the axis of the Lido Multi-Joint II System (Loredan Biomedical, Inc., Davis, California) exercise arm. Shin pad was positioned over the distal third of the lower extremity just above the malleoli. If the pad did not move up or down the lower extremity over the range of motion to be tested, the knee was considered to be aligned accurately with the axis of the exercise arm. The test range of motion (ROM) was set from full extension to 110° of flexion. Another pad positioned in lower third of thigh avoided movements in proximal lower extremity and a strap secured the pelvis. To ensure gravity-compensated torque values for the data analysis, the lower extremity was weighed by the Lido force transducer throughout full ROM.

Pairs of silver-silver chloride EMG surface electrodes (type Q-00-S, Medicotest, Olstykke, Denmark) were applied over the most prominent bellies of vastus lateralis, vastus medialis, rectus femoris and biceps femoris (long head). Center to center distance was 40 mm. A common ground electrode was placed apart in a electrically indifferent area. Raw EMG signal was amplified, full wave rectified, and smoothed with a low pass filter at a time constant of 12 msec and bandwidth of 10 to 1000 Hz using Mega EMG Station MESPEC 4001 (Mega Electronics Ltd., Kuopio, Finland).

Test procedure
There was submaximal warm-up and familiarization trial before the actual test. Five-minute rest periods were observed after the warm-up. For the actual test, each subject was asked to perform as many as possible maximal extension-flexion cycles during 1-minute exercise at joint velocity of 250°s⁻¹.

Data management
EMG data were analyzed with the aid of specially designed PC software ME4001 EMG Analyze System v.1.21 (Mega Electronics Ltd., Kuopio, Finland). Raw EMG was studied with respect of "cross-talk" within synergists and between antagonistic muscles before analysis. Randomly changed phase between EMG of a muscle pair assured absence of "cross-talk". Only the EMG of constant velocity area of the joint movement was analyzed. Velocity was considered constant, if it remained within the ± 5% of prescribed.

In order to obtain a measure of antagonist coactivity the average EMG (µV) of the antagonist at each joint angle was normalized
with respect of the average EMG of the muscle at the same angle acting as agonist (i.e. vastus lateralis average EMG at 60 deg of knee flexion was normalized with respect to its average EMG during maximal effort knee extension, and so on for each angle studied). Coactivity calculations were performed with the interval of 10 deg. Individual datum of each subject was obtained by calculating average of the three randomly selected cycles from the first and last 10 sec of the record when unfatigued and fatigued muscles were compared. Average coactivity of a muscle was calculated as an arithmetical mean of the cycle.

Muscle fatigue was measured as a shift in median frequency (MF) of the myoelectric power density spectrum (MPDS) to the lower frequencies. MPDS as well as its MF were calculated with the aim of Fast Fourier Transform.

Statistical analysis
One-way analysis of variance was used to test differences between mean values. For all statistics, the level of significance chosen was \( p < 0.05 \).

Results

Unfatigued muscle
Biceps femoris muscle exhibited the highest \( (p < 0.001) \) and rectus femoris the lowest coactivation \( (p < 0.001) \) among the muscles studied in football players (Fig 1, Panel A) whereas in ice hockey players...
only the coactivation of the biceps femoris differed remarkably (p < 0.001) from others (Fig 1, Panel B). Group B had significantly higher

rectus femoris coactivation (p < 0.001) as to compared to the group A. On the contrary, group A had significantly (p < 0.01) higher level of the biceps femoris coactivation during terminal 30 deg of the knee extension than group B.

**Fatigued muscle**

Median frequency of the MPDS decreased (p < 0.001) of all studied muscles in both groups during the test (Fig 2). This decrease

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**Figure 1, Panel B.**
Average coactivity of the knee antagonist muscles in ice hockey players

**Figure 2.** Average fatigability of the knee muscles during 20 maximum extension-flexion cycles. Front columns represent data of ice hockey players
was most prominent in vastus lateralis muscles of both groups (p < 0.05). Decrease in MF of the vastus lateralis in group B was still greater than in group A (p < 0.05).

Ability to coactivate decreased when muscles became fatigued. In Fig. 3 (Panels A and B) have presented coactivaty levels of the

![Figure 3, Panel A. Average coactivity of the fatigued knee antagonist muscles in the football players](image1)

![Figure 3, Panel B. Average coactivity of the fatigued knee antagonist muscles in ice hockey players](image2)

antagonist muscles with respect to joint angle. Initial 20 deg of the knee movement was accompanied by lowered coactivity (p < 0.05) of the antagonist muscles as to compared unfatigued. In the middle of the knee movement only fatigued vastus lateralis muscles of the both groups exhibited lowered coactivity (p < 0.05). Coactivity of all antagonist muscles was also lower during the last 30 deg of the knee movement.
Average coactivity level of fatigued muscles was lower than it was in unfatigued muscles (p < 0.001). As the most fatigued muscle vastus lateralis exhibited almost two times lower coactivation than it was in unfatigued conditions. Changes in MF correlated well with the changes in average coactivity (correlation coefficient $r = 0.92 - 0.77$ (p < 0.001 - 0.01).

**Discussion**

Results of the present study indicated that antagonist muscle coactivation was substantially modified by the fatigue in subjects studied. Coactivity response was remarkably lower in fatigued muscles than in unfatigued ones. This effect of the fatigue was most prominent at the initial and terminal phases of the knee movement. Correlation analysis revealed that more fatigued muscles (vastus lateralis) loss more in its ability to generate opposing torque to the agonist muscles than less fatigued.

The reduction in opposing antagonist activity at the initial phase of the movement results in reduction of the opposing torque it imparts to the joint. It may facilitate the initial acceleration of the limb, which could be considered useful. Similar phenomenon has been described in an earlier study (Hagood et al. 1990) with respect to the increased joint velocity. It has been, however, suggested that this initial increase in antagonist muscle coactivation, as it was also observed in unfatigued muscles of this study, increases joint stiffness that secures a proper direction of the agonist forces (Osternig et al. 1986).

Drastical decrease of the antagonist muscle coactivity at the terminal part of the movement in muscle fatigue, observed in ice hockey players, may harmfully affect knee biomechanics. Increase in final phase of joint movement provides active break for the limb (Solomonow et al 1987, Hagood et al 1990). This active break was clearly observed in ice hockey players, when muscles were not fatigued. Similar sharp increase in hamstrings coactivation towards the end of knee extension has been described in sprinters (Osternig et al. 1986) that is most likely typical for the low H/Q ratio. This mechanism may be of great importance in ice hockey players when considering that in skating the final extension must be powerfully performed (Herzog et al. 1991). Described mechanism may be significantly useful for the knee to prevent hyperextension and possible damage to the anterior cruciate ligament and joint capsule. On the other hand it has been shown that
Joint laxity is closely associated with the decreased tone of the antagonistic muscles (Skinner et al. 1986, Steiner et al. 1986). Therefore hamstring exercise is clearly indicated for the ice hockey players to protect the intact anterior cruciate ligament joint capsule.

The hamstrings were considerably more active during knee extension than the quadriceps during flexion. This may have been due to the more powerful quadriceps (especially in ice hockey players) requiring greater antagonist coactivation to co-ordinate joint movement than that required by the hamstrings. It is also possible that the larger mass of quadriceps provided a greater viscoelastic effect than the hamstrings. Lestienne (1979) has been reported that this passive viscoelastie force also serves as a control of the agonist force. Vastus lateralis was the most fatigued among the muscles studied. Vastus lateralis is the most powerful knee extensor muscle (Herzog et al. 1991). Its relative involvement in knee extension is greater than in other knee extensor muscles.

Conclusions

Our results indicated that muscular fatigue decreased the ability of the knee antagonist muscles to coactivate. Absence of the dynamic break may lead to the increased laxity of the knee joint and increase the risk of ligament and joint capsule damage, especially during powerful knee extension in ice hockey players. Special training of the hamstrings muscle is therefore recommended for the ice hockey players.

References


Kokkuvõte

Põlve antagonistlike lihaste koaktiivsust võrreldi jalgpalli-(n=14) ja jäähokimängijatel (n=14) maksimaalse isokieetilise harjutuse ajal nii puhanud kui ka väsinud lihaste korral. Jäähokimängijate hamstring-lihase koaktiviteet oli langenud võrreldes jalgpalluritega eriti põlveantastuses lõppfaasis. Lihasväsimus langetab lihase koaktivatsiooni võimet, mistõttu suureneb risk põlve ristatisidemetega (eriti eesmuse ristatisideme) vigastuse tekkeks. Eriti puudutab see jäähokimängijaid, kellel on hamstring-lihase koaktiviteet langenud ka puhanud lihase korral. Seetõttu on viimastel eriti oluline treenida hamstring-lihast.
COMPUTING AEROBIC AND ANAEROBIC LOAD PERIODS FROM HEART RATE RECORDS IN RUNNING

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Investigations were carried out in Tartu University, Department of sports medicine, Ülikooli 18, Tartu, Estonia, and in University of Kuopio, Department of Physiology, Kuopio 70211, PL6, Kuopio, Finland. Main author: Toomas Karu, prof.M.D., Hurda 13a, Tartu, Estonia.

Abstract — Heart rate is the easiest measured physiological parameter which reflects the intensity of physical exercise. Portable device "Sport-tester" stores the heart rate values in its memory and they can be further processed in a personal computer (PC). In this work a new additional method for distance-running heart rate curve analysis is described. The Heart Rate to Intensity (HRI) transfer program measures the heart rate curve, and the Heart Rate to Lactate (HRL) program is able to show how many minutes the subject was at each predicted lactate (LA) concentration level during his training session. Both programs need information about the individual heart rate at the anaerobic threshold, which must be previously established in a special field- or laboratory test. The theoretical relationship between HR and blood lactate in the HRL program has a very high coefficient of correlation (r=0.99). Accuracy is greatest at lactate level of 3-8 mMol/l and it is about 1-2 heart rate units. The method developed help subjects to train at proper individually effective levels and protects against excessive load.

Key words — Aerobic threshold, Anaerobic threshold, Computer analysis, Heart rate, Lactate, Distance running.

Introduction

Heavy endurance exercises are always accompanied by high lactate levels in muscles and blood. Recent investigations, carried out in
several countries and theoretical considerations of Wasserman et al. (17) show the important role of lactic acid (LA) in the efficiency of work rate above the anaerobic threshold (AT). Lactate production serves as a feedback control of the imbalance between the O2 requirement and supply. Lactate acts directly in four mechanisms 1) the Bohr effect, 2) vasodilatation, 3) remote conversion of lactate to pyruvate and 4) anaerobic phosphagen production. When lactate production elevates, several synchronous signs occur in the energy metabolism and cardiorespiratory system. These signs have been described by several investigators as "a lactate threshold" (8,3), "ventilatory anaerobic threshold" (3,4), "combined anaerobic threshold" (2), "maximal lactate steady state" (5) or "respiratory compensation threshold" (RCT) (13). The physiological and biochemical bases of these phenomena lie in working muscles. Recently Smirnov (15) has described 20 metabolic support regimens for running type exercises. Seluyanov et al. (12) postulated that these findings are connected with the functional activity of different types of the muscle fibers, depending on the intensity of physical exercise.

These metabolic and physiological signs occur in certain, very individual points, when the work load is increased.

The most important point in these reactions is the "anaerobic threshold" (AT). A market acceleration of lactate accumulation begins around 4 mMol/l (1). Some authors (13) use a fixed 4 mMol/l as AT, Wasserman et al. (13) defines AT, that is just below the point, at which the linearity in the VE/V02 and VE/WR curves markedly disappears. One of the best AT markers is the end of the FEC02 plateau (end of isocapnic buffer period) in the stepwise rising exercise protocol. It is in a good concordance with the break point of the heart rate in the Conconi test and is always very close to 4 mMol/l LA (1). In everyday training practice it seems to be one of the most important intensity borderlines. Another important borderline is the so-called aerobic threshold which seems to be highly correlated to the power of the mitochondrial citrate cycle (coefficient of correlation with citrate synthetase is 0.85 by Aunola (1). This borderline is usually 20-22 heartbeats lower than the above mentioned AT. There are thus two good exercise intensity markers, but there is a problem, how to use them in field conditions.

This paper describes a new methodical approaches how to provide a summary on influence of the whole training session on an athlete.
Material and Methods

Subjects
Six athletes (aged 22 to 28), members of Estonian national teams in tracks and field volunteered participated in this study (Table 1).

Table 1. Personal data of experimental group.

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</tr>
</tbody>
</table>

A and E have won several times Estonian championships and C has won the second place at previous USSR Championship in 1990.

Experimental procedures
Every person was carefully instructed about every test-procedure., i.e. a laboratory incremental test protocol on a treadmill (Morgan) and a field test with 4x400 m running blocks. The laboratory test was performed to establish V02max and heart rate changes at fully standardized conditions. Respiratory measurements in laboratory were carried out by the Oxycon-2 equipment (Mijnhardt, Holland). Heart rate was recorded by the Sport-Tester model PE 3000 (Polar Electro, Finland). The field test had 5-6 running blocks, 4x400 m each, on the Tartu University stadium (surface: recortan). Each test was preceded by a 10 min light warm-up. The interval between every block was about 5 min. On the fourth minute after every block samples of finger-tip blood for LA estimation were taken. For the analyses of blood lactate Biochemica Boehringer Mannheim enzymatic kits were used. The heart rate was recorded by Sport-tester PE 3000. The beginning of counting the heart-rate into the Sport-tester’s memory was switched on when starting the first 4x400 m lap. The athlete himself made intermediate markers in the beginning and at the end of every block.

For every person a special graph was constructed where the steady-state HR values (last minute of every 4x400 block) were plotted against the LA values on the fourth min after each block. An example of such a graph is presented in Fig. 1.
The 4mMol/l point on the lactate curve was used as the anaerobic threshold markerpoint. The running speed increased from block to block. The increments were equal throughout the test but individually determined by the coaches on the basis of the subject's physical fitness. All the tests were carried out until volitional exhaustion. After the test the heart rate values were transferred by the Polar interface to the IBM compatible personal computer (AT, Fountain, U.S.A.). Computer processing of the heart rate values was performed by the software program packet (version 3.20 A, Dec., 1990, Polar Electro) and our new HR analysis software. For statistical analyses the Statgraph program packet was used on IBM AT PC.

**Computer programs**

Several new programs for heart rate processing were worked out.

1. **The Heart Rate to Intensity Transfer program (HRI program).** This program calculates the heart rate values, recorded by the Sport-Tester into the following intensity ranges (15):
   Max - maximal endurance (all heart rate values above AT);
   S - special endurance (all heart rate values between AT and AerT).
G - general endurance (all heart rate values below AerT).

<table>
<thead>
<tr>
<th>TRANSFER TIME BY INTENSITY ZONES (HRI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name: Kukk</td>
</tr>
<tr>
<td>Date: 06-30-170</td>
</tr>
<tr>
<td>Last AT: 170</td>
</tr>
</tbody>
</table>

MAX 17
E3 9
E2 5
E1 7
R 28

Figure 2. Printout of Heart Rate to Intensity transfer program.

The HRI-TAB program does fastly the same in a table form (Table 2).

<table>
<thead>
<tr>
<th>TRAINING TIME BY INTENSITY ZONES (HRITAB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name: Kukk</td>
</tr>
<tr>
<td>Date: 06-30-1994</td>
</tr>
<tr>
<td>Last AT: 170</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>%</th>
<th>Interv.</th>
<th>Frequency</th>
<th>Intens.</th>
<th>HR/Sum</th>
<th>Time</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>26</td>
<td>170:200</td>
<td>-----------</td>
<td>max</td>
<td>30.9</td>
<td>17.0 min</td>
<td>61.7</td>
</tr>
<tr>
<td>14</td>
<td>160:170</td>
<td>-----------</td>
<td>SII</td>
<td>1.57</td>
<td>10.0 min</td>
<td>25.1</td>
</tr>
<tr>
<td>8</td>
<td>150:160</td>
<td>-----------</td>
<td>SI</td>
<td>8.2</td>
<td>5.0 min</td>
<td>9.8</td>
</tr>
<tr>
<td>10</td>
<td>135:150</td>
<td>-----------</td>
<td>GII</td>
<td>10.0</td>
<td>7.0 min</td>
<td>8.0</td>
</tr>
<tr>
<td>42</td>
<td>50:135</td>
<td>-----------</td>
<td>GI</td>
<td>31.2</td>
<td>8.0 min</td>
<td>12.5</td>
</tr>
</tbody>
</table>

Training Intensity Units: 11.7
Training Amount Units: 9.6
TOTAL: 21.3
SUMS: Anaer = 26%  Aer = 76%  G = 52%  S = 22%  Max = 26%

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The individual heart rate at the anaerobic threshold must be previously established in a laboratory or field test. In this program the
special endurance zone can be divided to I and II by various borderlines, found simply by subtracting from AerT several heartbeats. Usually the middle point between AT and AeT is used.

The HR1 program recalculates the Sport-Tester curve (Fig.1) into a histogram where the HR values are distributed by intensity zones (in minutes) (Fig. 2).

Distribution of HR was measured in thousands, minutes, percents and training units. The training units are obtained by multiplying the HR zone sums by 2,3,4 and 5; first zone was not multiplied.

2. The Heart Rate to Lactate Transfer program (HRL program). By means of a statistical analysis an extremely strong coefficient of correlation between the individualized HR and LA values (r=0.99) was found, and a special computer program was made to use this relationship (Karu and Käärik, 9). The Sport-Tester's output values are need to calculate how much time an athlete spends during a training session at certain blood lactate concentration. The measuring base is 4 mMol/l for individual heart rate at AT.

The HRL program makes printouts in graphic (Fig.3) and table form (Table 3). An attempt was made to use a special "Training index". It was found by counting the HR amount in thousands in every distribution zone and then multiplying the sum by the zone index. Indexing was linear (2,3,4...8).

<table>
<thead>
<tr>
<th>TRAINING TIME BY LACTATE UNITS (HRL)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Name:</strong> Kukk <strong>SII= 10</strong></td>
</tr>
<tr>
<td><strong>Date:</strong> 06-30-170 <strong>STI = 10</strong></td>
</tr>
<tr>
<td><strong>Last AT 170</strong> <strong>GII= 15</strong></td>
</tr>
<tr>
<td>L10 <strong>0</strong></td>
</tr>
<tr>
<td>L9 <strong>2</strong></td>
</tr>
<tr>
<td>L8 <strong>2</strong> VO2max</td>
</tr>
<tr>
<td>L7 <strong>2</strong></td>
</tr>
<tr>
<td>L6 <strong>3</strong></td>
</tr>
<tr>
<td>L5 <strong>4</strong></td>
</tr>
<tr>
<td>L4 <strong>5</strong> AT</td>
</tr>
<tr>
<td>L3 <strong>2</strong> AeT</td>
</tr>
<tr>
<td>L2 <strong>1</strong></td>
</tr>
<tr>
<td>L1 <strong>42</strong></td>
</tr>
</tbody>
</table>

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Figure 4. Printout of "Heart Rate to Lactate" transfer program in graphic form (HRL).
Table 3. Printout of the HRL program in table form

<table>
<thead>
<tr>
<th>%</th>
<th>Interv.</th>
<th>Frequency</th>
<th>Lact</th>
<th>HR/sum</th>
<th>Time</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>182:183</td>
<td>**</td>
<td>L8</td>
<td>18.3</td>
<td>3.0 min</td>
<td>14.6</td>
</tr>
<tr>
<td>4</td>
<td>1179:1182</td>
<td>**</td>
<td>L7</td>
<td>18.8</td>
<td>3.0 min</td>
<td>13.9</td>
</tr>
<tr>
<td>5</td>
<td>176:179</td>
<td>***</td>
<td>L6</td>
<td>23.0</td>
<td>3.0 min</td>
<td>15.8</td>
</tr>
<tr>
<td>6</td>
<td>172:176</td>
<td>***</td>
<td>L5</td>
<td>27.9</td>
<td>4.0 min</td>
<td>14.0</td>
</tr>
<tr>
<td>9</td>
<td>168:172</td>
<td>****</td>
<td>L4</td>
<td>30.0</td>
<td>6.0 min</td>
<td>15.5</td>
</tr>
<tr>
<td>3</td>
<td>161:164</td>
<td>**</td>
<td>L3</td>
<td>13.3</td>
<td>2.0 min</td>
<td>4.0</td>
</tr>
<tr>
<td>3</td>
<td>161:164</td>
<td>**</td>
<td>L2</td>
<td>11.3</td>
<td>2.0 min</td>
<td>3.3</td>
</tr>
<tr>
<td>62</td>
<td>70:161</td>
<td>**********</td>
<td>L1</td>
<td>207.2</td>
<td>42.0 min</td>
<td>20.7</td>
</tr>
</tbody>
</table>

Training units: 9.9

Sum: 359.6

65.0 min

98.8

Tartu University

Results

The main purpose of this investigation was connected with establishing a theoretical basis of the HRL program.

For every athlete we got a heart-rate curve by the Sport-Tester and Polar-ELECTro software. This curve was of an interval-type. At every exercise period the second half of the curve was averaged to establish a steady-state level by software tools. These HR values were named "exercise HR". After every exercise (4X400 m) period LA was measured, this value was stated as "exercise LA". The exercise HR and LA values were plotted for every athlete on a special graphic (Fig.5).

By means of this graph AT was found, the AT was fixed as the point where the experimental HR curve crosses the LA 4 line. So for every LA concentration its experimental HR value was found. These values are on Fig. 4 below the experimental curve.

On the Fig. 4 the theoretical (model) HR curve was shown, too. The theoretical HR values for every LA unit are shown above the curve. Next, the experimental and theoretical HR values for every LA unit were in pairs placed into Table 4.
Figure 4. *Plot of experimental and theoretical curves of heart rate and lactate. Dotted line - experimental, solid line theoretical curves.*

In Table 4 at first the difference in the experimental and theoretical HR values for every athlete at several LA units was found. It must be pointed out that concordance between these values is relatively higher above the AT point and less below AT. This table shows us in every case very high individual coefficients of correlation ($r=0.99$) between the experimental and theoretical HR values (Fig. 4).

**Discussion**

Two original computer programs were worked out. One of them - the "Heart Rate Intensity" (HRI) program is theoretically relatively simple. The program allows everybody to use that kind of AT he prefers because the computer asks its value. Consequently, rightness of a final result is here more a problem of goodness of the testing protocol and the experience of the laboratory staff.

More difficult problem arise around the second program - "Heart Rate to Lactate" (HRL). If this program is used by endurance athletes, and they do not use too short exercise periods (less than 1-3 min.), this program works quite satisfactory. Here is the main question, how stable are these relations after a longer periods of exercise, for
example 1, 2, 3 hours of physical effort. Furthermore, it may be influenced by environmental, nutritional or other factors.

Studies with 30 min. lasting endurance exercises by Urhausen et al. (16) show very similar results to our investigation, and relatively steady relationships have been found even in well-trained walkers (13). Maassen and Busse (11) have, however, postulated that overtraining or muscle glycogen depletion may change this relationship at least after a special diet conditions. According to Janssen’s (8) very large studies using heart rate recorded by means of the Sport-tester, the risk of instability of the HR/LA relationship from a practical viewpoint is not so serious.

Another problem is HRAT variations. Hellwig et al. (7) have found very large interindividual variations of this index (166-203 b/min). We have seen only a 160-182 b/min variation. König et al. (10) investigated day-to-day variations of performance, and the coefficient of variation in this case did not exceed 4-5%. Most athletes, especially with long career in sports, had very low HRAT variations in their annual training’s cycle (2-3 b/min).

We have used in the HRL program 4 mMol/l as a measuring base. May be it is more correct to use individual values of ATLA. They may vary from 3 to 5 mMol/l (7). The 4 mMol/l is relatively close to ventilatory AT too. WE used it as the HR/LA relationship basis, and it gave a good concordance between experimental and theoretical curves of this relationship in the most frequently used LA and HR area among the top athletes studies here. If the individual anaerobic threshold values are used, the program accuracy is increased.

There are several questions which require further studies. For example, it should be clarified if programs developed, work in swimming, where conditions are quite different from running. Specific influence may be produced by heavy enviromental, and kinesiologic conditions e.g. in cross-country skiing. These sport events are now under investigation at our laboratory.

Members of Estonian national teams used described programs in their training since 1991 in track and field athletics (middle and long distance run), cross-country skiing (we analyzed by means of them HR during the international competitions), cycling and orienteering. Athletes and coaches benefitted from the fast, complex and clear for understanding printouts. It is now much easier to manage the training process. They give a good orientation for training aims. Programs allowed to summarize the training data for a desired periods of days.
Conclusions

1. Analytical computing programs for heart rate allowed to quantitate the individual training intensity in endurance events in field conditions in relation to the anaerobic threshold of the athlete.

2. If in a laboratory or field test the heart rate at the lactate 4mMol/l has been established, it is possible to get computed LA concentrations during a training session by means of the HRL program.

Acknowledgements

We are deeply grateful for help and advice to the Polar Electro OY, especially to vice director Raimo Siurua and to the coaches of the Estonian national teams in skiing Mati Alaver and in track and field athletics for very enthusiastic collaboration.

References


to give running speed based on the lactate performance curve in the field - step-test. Int J Sports Med 12, 118


15. Suomalainen valmennusoppi II.


60
Abstract — The aim of this study is to determine the usefulness and limitations of noninvasive thoracic electrical bioimpedance (TEB) measurements simultaneously with oxygen uptake measurements for the assessment of cardiorespiratory reserve capacities in patients with coronary heart disease (CHD). Graded multistage upright exercise was performed on a bicycle ergometer. 25 patients with coronary heart disease after myocardial infarction, coronary artery bypass operation, or coronary angiography, demonstrating a stenosis >50% of the coronary artery were compared with 24 age adjusted healthy nontrained men (HM). Noninvasive hemodynamic and oxygen uptake measurements were used for the continuous monitoring of changes in the stroke volume, the rate of oxygen uptake, and the peripheral hemodynamic changes during the incremental workload ergometer test. The arteriovenous oxygen content difference was calculated indirectly by Fick's formula. At peak exercise, the patients demonstrated a reduced oxygen consumption (13.8±4.7 vs. 25.6±6.2 ml/kg/min p<0.001), stroke index (79±26 ml vs. 61±29 ml p<0.05), and ventilatory equivalent (47.7±8.9 ml/l vs. 40.6±7.3 ml/l p<0.05) when compared with normal subjects. At all the steps of the bicycle test we have found significant differences in oxygen uptake between the groups of pts. with CHD and healthy men (p<0.01). The stroke and cardiac indices were not statistically lower in the CHD patients in comparison with healthy men, on the submaximal levels of the ergometer test. There were no distinct differences between the groups in the heart rate and arteriovenous oxygen content difference at any step of the stress test, whereas distinct differences could be seen between the groups in the total peripheral vascular resistance (TPR) at rest (p<0.05) and starting from the stress test level 1.5 W/kg.
where TPR was essentially higher in the patients' group (p<0.05). Compared with normal subjects during submaximal exercise, the patients demonstrated a decreased oxygen consumption during all the stress test, accompanied by a decreased ventilatory equivalent starting from the stress test level 1.0 W/kg and a decreased outflow volume velocity starting from the stress test level 1.5 W/kg. Increased total peripheral resistance was associated with a decelerated outflow velocity starting from the bicycle test level 1.5 W/kg. The increased arterio-venous oxygen difference could not ensure the necessary oxygen delivery in the working muscles, so the maximal work capacity in the patient group was significantly lower in comparison with the healthy men (p<0.001) Our method of noninvasive measurements of hemodynamics and gas exchange enable us to assess the cardiorespiratory reserves in patients with coronary heart disease.

Key words — bioimpedance, hemodynamics, noninvasive, oxygen uptake, thoracic

Introduction

One of the most appreciated indices for the quantification of the cardiovascular function is maximal oxygen uptake during exercise. This is demonstrated by the failure to increase oxygen uptake despite an increase in work rate. However, it is easy to assess it in sportsmen who are accustomed to heavy exercise but is difficult or impossible in patients with CHD because of their inability to maximal level work of the test, so it is necessary to determine the cardiovascular function capacity at submaximal levels of exertion. Previous studies have shown a poor correlation between peak aerobic capacity and rest ejection fraction and cardiac output (Benge et al 1980, Meiler et al 1987). Several studies have suggested that peripheral factors such as the ability to direct the regional blood flow to the exercising skeletal muscle may be important (Wilson et al. 1984, Sullivan et al. 1989). There used to be a general agreement that pulmonary factors are not important because patients with heart failure typically do not develop arterial desaturation during exercise (Rubin et al. 1982, Sullivan et al. 1989). Nevertheless, more recent studies suggest that pulmonary and peripheral vascular adaptation may be an important determinant of exercise intolerance (Kraemer et al. 1993), and decreased oxygen-haemoglobin binding may be one of the adaptive mechanisms in clinical circumstances where
oxygen transport is inadequate (Serzin et al. 1993). Therefore, despite the large number of clinical studies addressing this problem, the exact mechanisms of exercise intolerance in heart failure have not been completely characterized. In particular, the potential contributions of peripheral vascular and pulmonary factors in relation to indices of cardiac function have not been clarified. This study is an attempt to determine the cardiorespiratory reserve capacities and peripheral vascular factors in patients with coronary artery disease at submaximal stress test levels by noninvasive simultaneous measurements of central and peripheral hemodynamic changes, and oxygen uptake.

**Subjects and methods.**

The study population consisted of two groups: 25 male patients with coronary heart disease (age range 32-50 years, mean 42±5) after myocardial infarction, coronary artery bypass surgery or coronary angiography demonstrating obstruction or narrowing >50% of at least one epicardial artery, and 24 nontrained healthy men (age range 33-47, mean 40±7 years.) Exclusion criteria included any known pulmonary disease, peripheral vascular disease, degenerative joint disease etc.

The incremental upright exercise test was performed on a bicycle ergometer. The initial load of exercise was set at 0.5 W per kilogram body mass and was increased by 0.5 W/kg. Each load lasted 3 minutes. The patients continued the exercise until there were complaints of chest pain, serious arrhythmias, reaching the target heart rate (85% of age predicted maximal), extreme fatigue or dyspnea.

At rest and at the end of each step of the incremental exercise test, the waveforms of the first derivative of thoracic electrical bioimpedance change were recorded simultaneously by EKG and phonocardiogram. The method is based on the measurement of changes in thoracical electrical impedance during the flow of alternating current (50 kHz and 0.35 mA). TEB was performed with four stretchable band electrodes according to the method described by Kubicek et al. (1966) using an impedance cardiograph P4-02 Minsk. The distances between the two inner electrodes at the front and back were measured and the mean value was used for the calculation. Stroke volume was calculated according to Kubicek's formula (Kubicek et al. 1966). To improve the accuracy of the stroke volume the correction coefficient rho for hematocrit changes according to the equation of Takada et al. (1981) was used. Cardiac output (CO) was calculated as stroke volume multiplied by heart rate (SV x HR). Cardiac output and stroke volume
were indexed to body surface area and expressed as a cardiac index (CI) and a stroke index (SI). For the assessment of the left ventricular contractile function we calculated an index of outflow volume velocity (OF), dividing SV by ejection time. Arterial blood pressure was measured auscultatorily by mercury sphygmomanometer at each step. Total peripheral resistance (TPR) was derived from the mean systemic arterial blood pressure and cardiac output, not taking into account the mean right arterial pressure (Grossman 1986) for every step of exercise test. The resistance formula was multiplied by a conversion factor of 80 to obtain resistance units of dynes sec cm⁻¹.

Expired gases were analysed at rest until a steady state was achieved, and then continuously during the exercise and recovery phase. The subjects wore a mouthpiece with a low resistance valve and a low volume (<150ml) of dead space in the circuit. Oxygen uptake (VO₂), carbon dioxide production (VCO₂) and minute ventilation (Ve) were measured during the third minute of each work load. The mixed expired air from the Douglas bag was analysed for the oxygen and carbon dioxide concentration by a paramagnetic O₂ analyser and infra-red CO₂ meter. The analysers were calibrated using reference gases of known concentrations. Values VO₂ and VCO₂ were corrected for atmospheric pressure, laboratory temperature and water vapour pressure. Oxygen consumption was adjusted to body weight VO₂/kg (ml/min/kg). Ventilatory equivalent (Eq) was calculated as the ratio of oxygen consumed per litre of inspired air. Eq = VO₂/Ve (ml/l).

Arterio-venous oxygen content difference (AVD) was found indirectly using Fick's formula. AVD=VO₂/CO.

Data analysis
To determine abnormal exercise response in patients, variables for this group were compared with those in the normal subjects. Group comparisons were made by using the unpaired Student's t-test. A p value < 0.05 was considered statistically significant.

Results
At first we compared the oxygen uptake and hemodynamic conditions at rest between the groups of CHD and healthy nontrained men. These indices were measured in the sitting position on the veloergometer before the exercise test. Statistically lower oxygen uptake was found in patients with CHD at rest in comparison with healthy men (3.9±1.1 ml/kg/min vs. 5.1±1.4 ml/kg/min p<0.01), there were no
differences in the ventilatory amount (12.7±3.5 l/min vs. 14.4±3.7 l/min). For the assessment of myocardial pumping function we compared the stroke index, cardiac index and heart rate in the sitting position before the test. A statistically lower stroke index was found in patients with CHD in comparison with healthy men (37±15 ml/m² vs. 47±18 ml/m² p<0.05) and no differences in the cardiac index (6.1±2.1 l/m² vs. 7.1±2.2 l/m²) and heart rate (84±12 beats/min vs. 81±14 beats/min). There was difference in the total peripheral resistance at rest between the groups: TPR was significantly higher in patients with CHD (1623±715 dyn-s-m⁻⁵ vs. 1164±455 dyn-s-m⁻⁵p<0.05).

During exercise much more oxygen and carbon dioxide are exchanged than at rest. Heart rate, cardiac output and oxygen consumption increased significantly during exercise in both groups. With increasing work level, as the pulmonary blood flow increases, both minute ventilation and alveolar ventilation increase immediately; in this way the lungs transfer more oxygen carbon dioxide and keep pace with metabolic demands. Oxygen uptake was significantly lower in patients with CHD at all the steps of the test (Figure 1).

**OXYGEN UPTAKE AT BICYCLE TEST**

![Figure 1](image)

There was no significant difference at the level of 2 W/kg between the pts. with CHD and healthy men, but there were only 2 CHD patients, who could continue the exercise test at this level. For the indirect assessment of pulmonary diffusing capacity we used index of ventilatory equivalent as a measure of oxygen utilization from ventilation. (Figure 2)
Ventilatory equivalent was significantly lower at the submaximal level of the bicycle test (1.0 W/kg) in patients with CHD (p<0.05). These indices refer to an uneconomical oxygen consumption in CHD patients. Heart rate, cardiac index and stroke index at any the levels of the ergometer test did not differ statistically between groups. (Figure 3).

The above indicators confirm that increased metabolic demands of an exercising skeletal muscle are compensated by appropriate increases in the blood flow at low workload. But at higher load of the exercise test
(1.5W/kg) there was a significant difference in the outflow volume velocity (Figure 4)

![Outflow Volume Velocity at Bicycle Test](image)

that was lower in patients with CHD than in the control group (p<0.05). The increased total peripheral resistance was associated with a decelerated outflow velocity. In our study the total peripheral resistance was significantly higher in the patients with CHD than in the healthy men (at level 1.5 W/kg of the bicycle test). (Figure 5)

![Total Peripheral Resistance at Bicycle Test](image)
There were no distinct differences in AVD between the groups of CHD and healthy men at rest and at the veloergometer test because of a large unstability between the subjects (Figure 6).

**ARTERIO-VENOUS OXYGEN DIFFERENCE AT BICYCLE TEST**

![Graph showing arterio-venous oxygen difference at bicycle test](image)

The hemoglobin content and erythrocytes count did not differ between the compared groups in our study. The mean hemoglobin concentration was 148 g/ml in the healthy men and 150 g/ml in the pts. with CHD (Table 1).

Increased arterio-venous difference could not provide the necessary oxygen for the working muscles, so the maximal work capacity in the patient group was significantly lower in comparison with the healthy nontrained men ($p<0.001$) (Table 1).

<table>
<thead>
<tr>
<th></th>
<th>Healthy men</th>
<th>CHD</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max. work level (W/kg)</td>
<td>2.0±0.3</td>
<td>1.1±0.5</td>
<td>$p&lt;0.001$</td>
</tr>
<tr>
<td>Max. SI (ml/m²)</td>
<td>79±26</td>
<td>61±29</td>
<td>$p&lt;0.05$</td>
</tr>
<tr>
<td>Max. VO₂ (ml/kg/min)</td>
<td>25.6±6.2</td>
<td>13.8±4.7</td>
<td>$p&lt;0.001$</td>
</tr>
<tr>
<td>Max. AVD (ml/l)</td>
<td>124±47</td>
<td>114±24</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>40±7</td>
<td>42±5</td>
<td></td>
</tr>
<tr>
<td>Erythrocytes (10¹²/l)</td>
<td>5.0±0.4</td>
<td>4.7±0.5</td>
<td></td>
</tr>
</tbody>
</table>
Discussion

Cardiac output increases linearly with $O_2$ consumption in normal subjects with a slope of approximately six. Six litres of blood with normal hemoglobin concentration contain only 1.2 l $O_2$. Thus, a task requiring an $O_2$ consumption increase of 1 l/min (e.g., brisk walking) that is accompanied by an increase in cardiac output of 6 l/min leaves only 0.2 l $O_2$ in muscle venous blood. This will result in a venous $O_2$ saturation of 18% ($PO_2$ of about 10 mm Hg) in the blood supplying the muscle, assuming that the entire increase in cardiac output would perfuse the exercising muscle. From this analysis, it is clear that the circulation barely meets the $O_2$ requirements in exercising muscles even in normal subjects. An impaired $O_2$ transport due to a primary cardiac defect to meet the oxygen requirements at work will be accompanied by muscle tissue hypoxia and increased muscle lactate output at inappropriately low work rates (Wasserman K.1989).

In our study $VO_2$ at rest was decreased in patients ($p<0.05$) and was decreased at submaximal exercise levels also. It agrees with Sullivan et al data (1989) where $VO_2$ at rest and at submaximal exercise levels were slightly decreased in patients with chronic heart failure. Our results show a significantly lower SI in CHD patients than in healthy men at rest that may indicate a damaged myocardial function in these patients.

Several reserve mechanisms come into play during the bicycle test in patients with left ventricular dysfunction. These include: increase of stroke volume via an increase in the left ventricular end-diastolic volume, and chronotropic responsiveness - tachycardia. We did not find any differences in stroke and cardiac indices during the whole of the ergometer test between the patients with CHD and healthy men, except a lower outflow velocity at the level of 1.5 W/kg that may show a decreased efficiency of the myocardial contractility.

The increased total peripheral resistance was associated with decelerated outflow velocity at the work level of 1.5W/kg. At exercise there occurs a redistribution of the systemic circulation, including vasodilatation in the skin and working muscles and vasoconstriction in the visceral organs and nonworking muscles. The net effect of vascular redistribution is a decrease in the systemic vascular resistance. An excessive vascular resistance may further restrain cardiac performance and the delivery of blood to exercising structures during exhaustive exercise (Meiler et al. 1987). The results of Wilson et al. (1984)
indicate that impaired nutritive flow to skeletal muscle is most likely the principal factor responsible for the reduced maximal exercise capacity of patients with heart failure.

Apart from increased cardiac output and vascular redistribution, a third mechanism to meet oxygen requirements is increased oxygen extraction from the arterial blood: this results in an increased arterio-venous content difference.

The pulmonary circulation also increases immediately with exercise. Unperfused alveoli become perfused (via recruitment of the pulmonary capillaries), and underperfused units receive an increased blood supply, and as a result, both the pulmonary blood volume and the pulmonary diffusing capacity for oxygen increase.

Limitations of this study.
This study is limited by the fact that the medical therapy was not standardized for all the patients. It is possible that some of the indices may have been affected to a greater degree than others by the previous medications. Also, the method has some technical limitations on this method. Since the monitoring of cardiac output is very sensitive to motion artefacts we used extendable electrodes and only one electrode around the neck (the other was around the head). During the recording of the bioimpedance waveform the exercise was interrupted for 5-10 sec. To exclude the effect of respiration five TEB complexes at every load of the stress test were measured and then averaged. A careful graphic analysis of the impedance signals remains essential for the validity of the method.

Summary

The results of these measurements in patients with coronary heart disease and in healthy men show the applicability of TEB and oxygen uptake for the estimation of cardiorespiratory reserves. We found essential differences in the peripheral resistance, ventilatory equivalent and outflow volume velocity at submaximal levels of exercise between the two groups studied. These data confirm the multifactorial etiology of exercise intolerance in patients with coronary heart disease. The pumping function of the myocardium is not the only factor limiting the physical work capacity in CHD patients: the disturbances in the efficiency of external respiration and peripheral resistance seem to play an important role. Despite of lower work capacity in CHD patients, AVD maximum did not differ between the
groups. We can conclude that in the case of a damaged myocardial function the O₂ supply is not compensated either by the increase of the O₂ appropriation in the lungs nor by the increase of the O₂ utilization in muscle cells.

This method of noninvasive measurements of hemodynamics and gas exchange can be used for continuous monitoring of cardiac performance without any significant risk and at low expense. We conclude that the measurements of respiratory gas exchange and central hemodynamics during exercise is an objective, reproducible and safe noninvasive method for characterizing the cardiac reserve and functional status in patients with coronary heart disease.

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Kokkuvõte

Uuringu eesmärgiks oli mitteinvasiivse torakaalse elektrilise
bioimpedants uuringu ja hapnikutarbimise üheaegse määramise
võimaluste selgitamine südame isheemiatõve haigetel (MIC)
kardiorespiratoorsete reservide määramiseks. Kasutati astmeliselt
tõusvate koormustega veloergomeetertersti. Uuriti 25 südame isheemiatõve
haiget, kes olid läbi põdenud südamelihase infarkti, läbi teinud
aorto-koronaarse šunteerimise või koronaarangiograafia, kus leitud
>50% stenoos koronaararteris. Võrdlusgrupiks kasutatud 24 mitte-
treenitud tervet meest vastavas vanuses.Kasutasime mitteinvasiivset
hemodünaamika ja hapnikutarbimise pidevat registreerimist lõögi-mahu,
hapnikutarbimise ja perifeersete hemodünaamika muutuste jälgingiseks
tõusvate koormustega veloergomeeterterstil. Arteriovenoosne hapniku
differents määratud kaudselt Ficki valemi abil.
Maksimaalsel koormisel oli haigetel oluliselt väiksem
hapnikutarbimine (13.3±4.7 ml/kg/min vrd. 25.6±6.2 ml/kg/min
p<0.001), lõogiindeks (79±26 ml/m² vrd. 61±29ml/m² p<0.05) ja ventilatsiooni-ekvivalent (47.7±8.9 ml/l vrd. 40.6±7.3 ml/l p<0.05) võrreldes tervetega. Koormutest igal astmel oli jälgitav oluline erinevus hapniku-tarbimises haigete ja tervete grupi vahel (p<0.01). Lõögi- ja südameindekseid ei olnud MIC haigetel tervetest statistiliselt tõepäraselt madalamad submaksmimaalsetel koormustel. Olulist erinevust uuritud gruppide vahel ei ilmnunud ei pulsisageduses, ventilatsiooni mahus ega hapniku arterio-venooses differentsis. Tõepärane erinevus ilmes totalises perifeerises takistuses alates koormusest 1.5W/kg, mis oli oluliselt kõrgem haigete grupis (p<0.05). Tervetega võrreldes ilmes haigetel submaksmimaalsetel koormustel väiksem hapnikutarbimine kogu koormuse jooksul, millega kaasnes ventilatsiooniekvivalendi langus alates koormusest 1.0W/kg ja väljutuse mahtkiiruse langus alates koormusest 1.5W/kg, viimasega kaasnes totaluse perifeerise takistuse tõus koormusel 1.5W/kg. Hapniku arterio-venooses differentsi tõus ei kindlutanud vajalikku hapnikuga varustamist töötava lihastes, seetõttu on haigete grupi maksimaalne töövõime tervetega võrreldes oluliselt madalam (p<0.001).

Kasutatav mitteinvasiivne hemodünaamika ja gaasdivahetuse määramise metoodika võimaldab edukalt hinnata koronaartöve haigete kardiorespiratoorseid reserve.
Abstract — In this study a system of standards for evaluating physical capacity indices in men of different age is presented. In the study 312 men aged 21-60 years participated. We measured their weight, height, body mass index and main physical capacity indices: strength, flexibility, skill, endurance and speed. Most of the indices had fixed age-related dynamics and we offer 2 systems for evaluating the indices: a) if there is any dynamics in indices with age the indices of the two groups are compared (below/over 40 years old; b) if there is no dynamics with age, the standard is given as for one group (21-60 years old). To evaluate physical capacity indices, 6 grades starting from "very poor" to "excellent" were used. These standards can be used for evaluating physical capacity indices and also for comparing changes during training or disease preventing programs.

Key words — morphological capacity, physical capacity, standards

Introduction

Recently the Estonian population has showed a greater interest in its health, particularly in the positive side of their health: how high is their physical work capacity, how good is their tolerance to physical load, how fast are their recovery processes and how their physical capacities are developing. To answer these questions one should know the standard for a definite age and sex. In Estonia the standards for youth have been worked out (Silla, Teoste, 1989). For adults so far we only have standards for evaluating maximal oxygen consumption (Pärnat, 1982).

The aim of the study was to determine and analyse morphologic and physical capacity indices in men and to work out standards for men of different age groups.
**Subjects and Methods**

In the Department of Sports Medicine and Rehabilitation 312 men aged 21-60 were investigated. Morphologic indices like weight and height were determined and relative body weight (body mass index, BMI) was calculated. To calculate BMI, weight (kg) is divided by height squared (m²). We determined the following physical capacities:

1) muscular strength. Strength was measured by the hand-grip test.
2) flexibility. Flexibility was determined by the extent of a maximal downward bending.
3) skill. Skill was evaluated on the basis of 3 exercises needing coordination and balance skills.
4) endurance. Endurance was determined in 2 ways:
   a) by using the veloergometric test where the participants had to work with load 100 W during 3 minute period (König et al., 1966). We determined the heart rate (HR) and blood pressure (BP) before the test, at the 3rd minute of the test and at 3rd minute after the test.
   b) by using the step-test (Amossov, 1985). The participants had to walk slowly from the ground to the 5th floor. We measured the time spent on covering the distance. Heart rate and blood pressure were measured before and after the test.

5) speed. We determined 3 kinds of speed:
   a) reaction speed. Reaction speed was determined by Dushanin's method (Dushanin 1985).
   b) speed of motion. The participants ran at maximal speed from the ground to the 5th floor. We measured the time to complete the distance, and the heart rate and blood pressure before and after the test.
   c) speed of movements. The participants had to make maximal amount of revolutions on bike ergometer during 10 s at load of 100 W.

The results were processed statistically, we carried out correlation and regression analyses and to compare different indices ANCOVA was used.

**Results and Discussion**

The results of the study give us the conception of the direction and the character of the age-related changes in the indices of men and could serve as the basis for creating standards.

The means of morphological indices, skill, flexibility and reaction speed have definite dynamics with age (Table 1).
In all age groups the individual values varied in a wide range. This fact shows that there must be standards for each index in all age groups.

The results of the veloergometric test show smaller dynamics of age-related changes (Table 2). To evaluate the character of an organism's adaptation to load we need to determine the reaction type to physical load. The amount of atypical reaction types to increases with age and hypertonic reaction type to physical load occurs more frequently.
Table 2. Results of the veloergometric test in men (Mean±SD)

<table>
<thead>
<tr>
<th>No.</th>
<th>Index</th>
<th>21-30</th>
<th>31-40</th>
<th>41-50</th>
<th>51-60</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>n</td>
<td>69</td>
<td>76</td>
<td>99</td>
<td>68</td>
</tr>
<tr>
<td>2.</td>
<td>Heart rate (bpm)</td>
<td>78.4±12.5</td>
<td>78.3±11.3</td>
<td>78.9±11.7</td>
<td>81.4±15.4</td>
</tr>
<tr>
<td>3.</td>
<td>Blood pressure syst. (mm Hg)</td>
<td>127.1±12.6</td>
<td>131.5±14.5</td>
<td>129.7±17.7</td>
<td>134.5±18.5</td>
</tr>
<tr>
<td>4.</td>
<td>Blood pressure diast. (mm Hg)</td>
<td>82.0±9.7</td>
<td>84.0±9.8</td>
<td>85.2±11.1</td>
<td>87.7±11.2</td>
</tr>
<tr>
<td>5.</td>
<td>Heart rate at 100W (bpm)</td>
<td>118.4±18.4</td>
<td>125.7±19.6</td>
<td>115.5±19.9</td>
<td>119.6±18.2</td>
</tr>
<tr>
<td>6.</td>
<td>Blood pressure syst. at 100W</td>
<td>148.8±18.9</td>
<td>154.1±19.1</td>
<td>158.3±25.3</td>
<td>159.6±20.3</td>
</tr>
<tr>
<td>7.</td>
<td>Blood pressure diast. at 100W</td>
<td>79.6±12.3</td>
<td>82.9±12.7</td>
<td>87.2±12.7</td>
<td>91.3±12.1</td>
</tr>
<tr>
<td>8.</td>
<td>Revolutions (n)</td>
<td>23.5±3.5</td>
<td>22.6±4.0</td>
<td>21.1±3.1</td>
<td>20.7±4.5</td>
</tr>
<tr>
<td>9.</td>
<td>Recovery heart rate 3' (bpm)</td>
<td>84.8±16.5</td>
<td>91.9±13.6</td>
<td>86.6±14.4</td>
<td>88.8±13.9</td>
</tr>
<tr>
<td>10.</td>
<td>Recovery blood pressure syst 3'</td>
<td>141.0±14.6</td>
<td>133.5±15.6</td>
<td>134.5±16.5</td>
<td>137.1±18.6</td>
</tr>
<tr>
<td>11.</td>
<td>Recovery blood pressure diast. 3'</td>
<td>78.9±11.4</td>
<td>83.6±11.8</td>
<td>83.7±13.0</td>
<td>88.3±13.1</td>
</tr>
</tbody>
</table>

The changes in the heart rate and blood pressure during the step-tests (Tables 3,4) are more expressed because the step-tests were harder than the veloergometric test. Also the step-test determining speed was harder than the step-test determining endurance. Most of the indices show statistically significant differences between age groups. The greatest changes between the age groups occur in speed of motion. The amount of atypical reaction types (hypertonic and hypotonic) increased with age, especially at the step-test determining speed.

Correlation analysis showed that between similar indices at different stages of the tests the coefficient of correlation was quite high (BP syst. before ↔ BP syst at 100W - r=0.75). Correlation was rarely found between indices that evaluate different morphological and physical capacities. (Age ↔ BP syst. - r=0.50). Hence, it is necessary to carry out complex investigations of an organism.

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Table 3. Results of the step-test determining endurance (Mean±SD)

<table>
<thead>
<tr>
<th>No</th>
<th>Index</th>
<th>Age</th>
<th></th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>21-30</td>
<td>31-40</td>
<td>41-50</td>
<td>51-60</td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>n</td>
<td>66</td>
<td>70</td>
<td>89</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Heart rate (bpm)</td>
<td>74.3±</td>
<td>74.3±</td>
<td>78.7±</td>
<td>83.1±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.0</td>
<td>9.8</td>
<td>11.4</td>
<td>15.4</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Blood pressure syst. (mm/Hg)</td>
<td>127.2±</td>
<td>128.0±</td>
<td>137.5±</td>
<td>138.4±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±11.5</td>
<td>12.0</td>
<td>15.4</td>
<td>15.8</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Blood pressure diast. (mm/Hg)</td>
<td>81.1±</td>
<td>82.5±</td>
<td>88.6±</td>
<td>88.6±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.1</td>
<td>12.1</td>
<td>11.4</td>
<td>11.1</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Time (sec.)</td>
<td>64.2±</td>
<td>67.6±</td>
<td>71.4±</td>
<td>75.5±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>17.2</td>
<td>11.7</td>
<td>16.7</td>
<td>14.4</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Recovery heart rate 1' (bpm)</td>
<td>124.2±</td>
<td>113.9±</td>
<td>113.1±</td>
<td>118.3±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±17.6</td>
<td>20.1</td>
<td>16.7</td>
<td>20.7</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Recovery blood pressure syst. 1'</td>
<td>158.4±</td>
<td>158.9±</td>
<td>163.1±</td>
<td>168.8±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±18.2</td>
<td>22.5</td>
<td>22.4</td>
<td>30.4</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Recovery blood pressure diast. 1'</td>
<td>81.7±</td>
<td>90.4±</td>
<td>88.6±</td>
<td>90.3±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.3</td>
<td>14.4</td>
<td>15.6</td>
<td>11.2</td>
<td></td>
</tr>
</tbody>
</table>

Table 4. Results of the step-test determining speed (Mean±SD)

<table>
<thead>
<tr>
<th>No</th>
<th>Index</th>
<th>Age</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>31-40</td>
<td>41-50</td>
<td>51-60</td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>n</td>
<td>58</td>
<td>66</td>
<td>71</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Heart rate (bpm)</td>
<td>78.5±</td>
<td>84.0±</td>
<td>86.4±</td>
<td>89.2±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>12.3</td>
<td>12.0</td>
<td>14.8</td>
<td>19.8</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Blood pressure syst. (mm Hg)</td>
<td>133.7±</td>
<td>136.6±</td>
<td>145.0±</td>
<td>145.2±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±13.5</td>
<td>14.5</td>
<td>19.2</td>
<td>15.3</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Blood pressure diast. (mm Hg)</td>
<td>82.6±</td>
<td>84.7±</td>
<td>90.7±</td>
<td>88.2±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.1</td>
<td>7.6</td>
<td>13.2</td>
<td>9.3</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Time (sec.)</td>
<td>28.7±</td>
<td>29.9±</td>
<td>34.9±</td>
<td>42.9±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>13.4</td>
<td>4.6</td>
<td>6.4</td>
<td>14.3</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Recovery heart rate 1' (bpm)</td>
<td>149.4±</td>
<td>135.9±</td>
<td>133.9±</td>
<td>137.3±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±17.6</td>
<td>19.2</td>
<td>18.6</td>
<td>24.1</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Recovery blood pressure syst. 1'</td>
<td>178.2±</td>
<td>171.4±</td>
<td>171.3±</td>
<td>182.7±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±19.8</td>
<td>21.9</td>
<td>22.3</td>
<td>31.9</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Recovery blood pressure diast. 1'</td>
<td>78.7±</td>
<td>86.4±</td>
<td>85.2±</td>
<td>87.2±</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.7</td>
<td>14.3</td>
<td>14.4</td>
<td>14.4</td>
<td></td>
</tr>
</tbody>
</table>
At first standards for evaluating single indices were worked out: for maximal oxygen consumption (Astrand, Rodahl, 1970), vital capacity (Vasar, Laidre, 1974), anthropometric indices (Aul, 1974), aerobic capacity (Cooper, 1979) etc. Later a complex of indices was used to work our standards for evaluating an organism's functions (Dushanin et al., 1985; Prevarski, Butkevitch, 1985; Pirogova et al., 1986; Apanasenko, 1989; Jürimäe, 1990; Kurnosov, 1992). It is difficult to use these systems because each author has his own selection of indices which have been influenced by geographical and ethnic factors.

First standards were worked out as expert assessments. As an example we present the standards for evaluating the relative body weight (BMI) which can be applied to men of any age (Table 5).

**Table 5. Standards for evaluating relative body weight**

<table>
<thead>
<tr>
<th>BMI</th>
<th>EVALUATION</th>
<th>OVERWEIGHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;19</td>
<td>Underweight</td>
<td>-</td>
</tr>
<tr>
<td>19-24</td>
<td>Proper weight</td>
<td>-</td>
</tr>
<tr>
<td>25-27</td>
<td>Slight overweight</td>
<td>1-10%</td>
</tr>
<tr>
<td>28-30</td>
<td>Moderate overweight</td>
<td>11-20%</td>
</tr>
<tr>
<td>31-40</td>
<td>Significant overweight</td>
<td>21-60%</td>
</tr>
<tr>
<td>&gt;40</td>
<td>Severe overweight</td>
<td>over 60%</td>
</tr>
</tbody>
</table>

As a mathematical method for working out standards mean and standard deviation were used and this enabled us to divide values into 5 classes. This method can be applied to indices with normal distribution. To indices with asymmetric distribution the system of median and its relation to maximum $[(median \Rightarrow maximum)/3]$ and minimum $[(minimum \Leftarrow median)/3]$ values (Karu, 1976) were used. This system allows us to have 6 different gradations: from "very poor" to "excellent". As we divided the participants into groups of the same decades of life, we could trace the dynamics of age-related changes, but in many cases the difference between the age groups was statistically non-significant. In addition we compared of indices in men under 40 and over 40 years old. We discovered statistically significant changes in most indices between both groups, only in 3 indices no statistically significant changes were found. Taking all that into account standards for evaluating the indices were worked out (Tables 6,7).
Table 6. Standards for evaluating strength, skill and flexibility in men.

<table>
<thead>
<tr>
<th>Index</th>
<th>Age (years)</th>
<th>Very poor</th>
<th>Poor</th>
<th>Fair</th>
<th>Average</th>
<th>Good</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand-grip dex. (kg)</td>
<td>&lt;40</td>
<td>&lt;45</td>
<td>45-54</td>
<td>55-64</td>
<td>65-69</td>
<td>70-74</td>
<td>&gt;74</td>
</tr>
<tr>
<td></td>
<td>&gt;40</td>
<td>&lt;35</td>
<td>35-44</td>
<td>45-54</td>
<td>55-59</td>
<td>60-64</td>
<td>&gt;64</td>
</tr>
<tr>
<td>Hand-grip sin. (kg)</td>
<td>&lt;40</td>
<td>&lt;43</td>
<td>43-52</td>
<td>53-62</td>
<td>63-67</td>
<td>68-72</td>
<td>&gt;72</td>
</tr>
<tr>
<td></td>
<td>&gt;40</td>
<td>&lt;33</td>
<td>33-42</td>
<td>43-52</td>
<td>53-57</td>
<td>58-63</td>
<td>&gt;63</td>
</tr>
<tr>
<td>Flexibility (cm)</td>
<td>&lt;40</td>
<td>&gt;20</td>
<td>20-16</td>
<td>15-11</td>
<td>10-6</td>
<td>5-1</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td>&gt;40</td>
<td>&gt;25</td>
<td>25-21</td>
<td>20-16</td>
<td>15-11</td>
<td>10-6</td>
<td>&lt;6</td>
</tr>
<tr>
<td>Skill (mark)</td>
<td>21-60</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Table 7. Standards for evaluating speed and endurance indices in men.

<table>
<thead>
<tr>
<th>Endurance</th>
<th>GRADE</th>
<th>Age (years)</th>
<th>Very poor</th>
<th>Poor</th>
<th>Fair</th>
<th>Average</th>
<th>Good</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm) (veloergo 100W)</td>
<td>21-60</td>
<td>&gt;150</td>
<td>150-136</td>
<td>135-121</td>
<td>120-111</td>
<td>110-100</td>
<td>&lt;100</td>
<td></td>
</tr>
<tr>
<td>Time (sec) (step-test)</td>
<td>&lt;40</td>
<td>&gt;100</td>
<td>100-86</td>
<td>85-71</td>
<td>70-61</td>
<td>60-51</td>
<td>&lt;51</td>
<td></td>
</tr>
<tr>
<td>Time (sec) (step-test)</td>
<td>&gt;40</td>
<td>&gt;110</td>
<td>110-96</td>
<td>95-81</td>
<td>80-71</td>
<td>70-61</td>
<td>&lt;61</td>
<td></td>
</tr>
<tr>
<td>Speed of movements (sec.)</td>
<td>21-60</td>
<td>&lt;16</td>
<td>16-18</td>
<td>19-21</td>
<td>22-25</td>
<td>26-29</td>
<td>&gt;29</td>
<td></td>
</tr>
<tr>
<td>Reaction speed (cm)</td>
<td>&lt;40</td>
<td>&gt;30</td>
<td>30-26</td>
<td>25-21</td>
<td>20-17</td>
<td>16-12</td>
<td>&lt;12</td>
<td></td>
</tr>
<tr>
<td>Reaction speed (cm)</td>
<td>&gt;40</td>
<td>&gt;50</td>
<td>50-38</td>
<td>37-26</td>
<td>25-21</td>
<td>20-16</td>
<td>&lt;16</td>
<td></td>
</tr>
<tr>
<td>Speed of motion (sec.)</td>
<td>&lt;40</td>
<td>&gt;50</td>
<td>50-41</td>
<td>40-31</td>
<td>30-28</td>
<td>27-25</td>
<td>&lt;25</td>
<td></td>
</tr>
<tr>
<td>Speed of motion (sec.)</td>
<td>&gt;40</td>
<td>&gt;60</td>
<td>60-51</td>
<td>50-41</td>
<td>40-36</td>
<td>35-31</td>
<td>&lt;31</td>
<td></td>
</tr>
</tbody>
</table>
Conclusion

On the basis of the data obtained as a result of investigation of 312 men of different age standards for evaluating main physical capacity indices were worked out. These standards can be used in clinical and preventive medicine for evaluating the current condition of an organism and to compare changes developing as a result of training and disease preventing programs.

References


10. Jürimäe T (1990) The scale for evaluating physical work capacity in the students of Tartu University. Tartu (in Estonian)


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Kokkuvõte

Tänapäeval tuntakse üha enamat huvi oma tervise vastu. Eelkõige huvitab kõiki tervise positiivne külg: kui suur on organismi töövõime, kuidas arenevad inimene kehalised võimed, kui hea on tema koormustaluvus või kui kiired on temal taastumisprotsessid. Nende küsimustele vastamiseks on vaja teada ealisi ja soolisi standardeid.

Uuringu eesmärgiks oli ealiste standardite väljatöötamine Eesti meeste jaoks. Uuringu käigus uurisime 312 meest vanuses 21-60 aastat ning saime andmeid nende morfooloogiliste näitajate ja organismi põhiliste kehaliste võimete (jõud, painduvus, osavus, kiirus, vastupidavus) kohta. Hindasime ka organismi adaptatsiooni koormussele.

Saadud andmepank lubas välja töötada standardid kehaliste võimete hindamiseks. Enamikku näitajatest hindasime vanusegruppide järgi (alla 40 a./üle 40 a.). Näitajate puhul, kus ealist dünaamikat ei täheldatud, toimus hinnang ühes vanusegrupis (21-60 a.)

Väljatöötatud standardid võib kasutada kehaliste võimete hindamiseks meestel aga ka muutuste dünaamika hindamisel treeningprogrammide ja tervistavate programmide täitmisel.
FITNESS, PHYSICAL ACTIVITY AND SERUM CHOLESTEROL OF MEN OF DIFFERENT AGE GROUPS

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Abstract — Fitness, physical activity and serum cholesterol of men in Estonia have become a medical and social problem. Men fall ill with cardiac diseases more frequently and mortality from these diseases is higher than in women in Estonia. Physical activity, fitness, morphological and laboratory indices of men of different age groups were determined in this study. In the study participate 209 men aged 17-67 years. Most of the participants deal with sedentary and light physical work and have a low physical activity. It leads to an increase in weight and body fat. Serum cholesterol in blood is elevated in many participants in all age groups. This calls forth changes in adaptation to physical loads and the amount of hypertonic reactions to physical load increases significantly. Physical work capacity and maximal oxygen consumption decrease slightly with age. Changes in ECG during the physical load increase significantly with age. The data of the study allow to prescribe physical activity, diet and changes in lifestyle.

Introduction

Diseases of the cardiovascular system are one of the major adult health problems and reason of premature death in Estonia (Jänes, 1992; Leinsalu, 1993). The main risk factors are assessed to be general stress and depression in connection with destability in political, economical and psychological situation, mistakes in dietary practices (more often overnutrition), smoking, hypertension and low physical activity (Criqui et al., 1980; Hockey, 1981; Kaplan, 1989).

The aim of the study was to examine the work characteristics and physical activity and to determine morphologic, laboratory and fitness indices of men of different age groups.
Subjects and Methods

We examined 209 men aged 17-67 years. The subjects were divided into 2 age groups (below 40 and over 40 years old) to analyse their work characteristics and physical activity. Our questionnaire contained questions on general, medical and sports anamnesis and on risk factors of coronary heart disease. To analyse the laboratory, morphologic and fitness indices of the participants they were divided into groups of the same decades of life (under 21, 21-30, 31-40, 41-50, 51-60, over 60 years old). We determined the following morphological indices: weight, height, vital capacity and body fat (Parizkova, 1977). We established haemoglobin, sedimentation (ST), serum cholesterol, α-lipoproteid, glucose and erythrocytes in blood. The amount of serum cholesterol in blood was determined by the Lieberman-Burchard reaction, the amount of α-lipoproteid by the Burstain method and the amount of glucose by orototoluidine method. Physical work capacity (PWC$_{170}$) was tested on bicycle ergometer using increasing loads. The first load was 50 W within 3 minutes, the second 75, 100, 125, 150 or 200 W according to the heart rate during the first load. Maximal oxygen consumption (VO$_{2\text{max}}$) was calculated using nomogram (Karpman et al., 1988). Electrocardiogram (ECG), heart rate (HR) and blood pressure (BP) were registered before, during and after the ergometer test. The averages, standard deviations and standard errors of the variables were calculated. We also carried out correlation and regression analyses, and, to find the association between different indices, Student's t-test was used.

Results and Discussion

The presented data about the work characteristics (Fig 1.) depict the current situation in a modern society. In both groups the participants occupied in sedentary and light work prevail. The number of people dealing with moderate and hard physical work is insignificant. Such a tendency will persist or even increase in the nearest future.
The difference of work characteristics of men between the age groups was insignificant. About a quarter of the participants do not deal with leisure-time training at all. Although the number of participants training 1-2 times a week is relatively high (38.4% in the "younger" group and 40.0% in the "older" group), the effect of these trainings is low. It is proved that in order to maintain or increase fitness one should train 3 or more times a week (Pollock et al., 1979; Shepard, 1986;
Vuori, 1988). Only one third of the participants train sufficiently. Data analysis shows that those who are occupied in moderate and hard physical work have greater physical activity at their leisure-time. Our parallel studies show that the percentage of Estonian women occupied in sedentary or light physical work and having insufficient physical activity is greater (Täll et al., 1994).

The problem of insufficient physical load should be solved by individual leisure-time training (Paffenberger et al., 1987; Chandrashekhar et al. 1991; Gordon et al., 1991). Recommended intensity of training is 60-90 % of maximum heart rate or 50-85 % of VO₂max, and the duration 20-60 minutes of continuous aerobic activity and during the activity large muscle groups should be involved, e.g., walking, jogging, cycling, swimming, cross-country skiing and rowing. Physical activity improves general adaptation mechanism, increases an organism’s resistance, reduces the amount of serum cholesterol and has anti sclerotic effect. Our data of physical activity at leisure-time are presented in Fig 2.

![Graph showing physical activity of men of different age groups](image.png)

**Figure 2. Physical Activity of Men of Different Age Groups.**

In order to analyse the morphologic and laboratory indices participants were divided into groups of the same decades of life (Table 1).
<table>
<thead>
<tr>
<th>No.</th>
<th>Age (years)</th>
<th>n</th>
<th>Length (cm)</th>
<th>Weight (kg)</th>
<th>Vital capacity (l)</th>
<th>Body fat (%)</th>
<th>Hemoglobin (g/l)</th>
<th>ST (mm/Hg)</th>
<th>Serum cholesterol (mmol/l)</th>
<th>Glucose (mmol/l)</th>
<th>( \alpha )-Lipoproteids (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>&lt;21</td>
<td>15</td>
<td>182.2±1.91</td>
<td>71.3±2.79</td>
<td>4.4±0.2</td>
<td>12.7±1.31</td>
<td>144.0±3.23</td>
<td>3.8±0.31</td>
<td>5.2±0.3</td>
<td>3.8±0.15</td>
<td>1.54±0.08</td>
</tr>
<tr>
<td>2.</td>
<td>21-30</td>
<td>54</td>
<td>181.4±0.85</td>
<td>78.7±1.4</td>
<td>4.7±0.1</td>
<td>16.5±0.74</td>
<td>149.2±2.03</td>
<td>4.8±0.53</td>
<td>5.95±0.15</td>
<td>4.01±0.1</td>
<td>1.53±0.04</td>
</tr>
<tr>
<td>3.</td>
<td>31-40</td>
<td>42</td>
<td>181.4±1.01</td>
<td>84.5±2.27</td>
<td>4.5±0.1</td>
<td>18.4±0.86</td>
<td>147.1±2.34</td>
<td>6.4±0.74</td>
<td>5.94±0.18</td>
<td>4.3±0.16</td>
<td>1.51±0.05</td>
</tr>
<tr>
<td>4.</td>
<td>41-50</td>
<td>54</td>
<td>179.9±0.96</td>
<td>83.7±1.67</td>
<td>4.4±0.1</td>
<td>19.2±0.6</td>
<td>145.9±2.07</td>
<td>5.8±0.48</td>
<td>6.12±0.15</td>
<td>4.4±0.13</td>
<td>1.55±0.04</td>
</tr>
<tr>
<td>5.</td>
<td>51-60</td>
<td>29</td>
<td>178.2±1.12</td>
<td>84.6±2.21</td>
<td>4.1±0.1</td>
<td>19.9±0.72</td>
<td>150.1±2.38</td>
<td>8.9±1.7</td>
<td>6.23±0.25</td>
<td>4.8±0.15</td>
<td>1.46±0.06</td>
</tr>
<tr>
<td>6.</td>
<td>&gt;60</td>
<td>15</td>
<td>174.1±1.7</td>
<td>77.2±3.21</td>
<td>3.3±0.1</td>
<td>18.9±1.21</td>
<td>148.1±3.89</td>
<td>7.1±1.31</td>
<td>6.94±0.35</td>
<td>4.5±0.27</td>
<td>1.72±0.07</td>
</tr>
</tbody>
</table>
The means of weight increase significantly due to the increase of body fat (r-0.51) with age. Biochemical indices confirm these changes: the amount of serum cholesterol in blood increases with age (r-0.41). Hypercholesterinemia can be found in all groups. The dynamics of hypercholesterinemia are depicted in Fig 3. Already at the age of 40 the means of serum cholesterol in blood exceeded 6.0 mmol/l. This situation significantly increases the possibility of developing the atherosclerotic processes (Bønaa et al., 1992; Witten, 1993). Analogous results have also been found by other authors (Marrugat, 1993; Nilson et al., 1993). The amount of α-lipoproteid in blood does not change significantly with age.

![Fig 3. Occurrence of Hypercholesterinemia in Men.](image)

Several authors have found that high physical activity reduces the level of serum cholesterol in blood (Brownell et al., 1982; Giada et al., 1991; Gordon et al., 1991). This fact should be used to finding the development of atherosclerotic processes in organism (Rauramaa et al., 1984; Thompson et al., 1988; Eaton, 1992). In our study the level of glucose in blood exceeded the limits only in a few cases.

The following data were obtained after the veloergometric test (Table 2). The means of the heart rate before the test were almost equal in all groups, but individual indices varied in quite a wide range. Heart rate correlates with atherogenic blood lipid. Persons participating in physical training have generally lower heart rate and their blood lipid levels differ from persons with high heart rate (Bønaa et al., 1992). We found it difficult to analyse the means of heart rate during the test because the load for each person was different. We would like to stress
Table 2. Results of the veloergometric test (M±m).

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (years)</th>
<th>n</th>
<th>Heart rate (bpm)</th>
<th>BP syst. (mm Hg)</th>
<th>BP diast. (mm Hg)</th>
<th>Test HR (bpm)</th>
<th>Test BP syst (mm Hg)</th>
<th>Test BP diast.</th>
<th>PWC(_{170}) (W)</th>
<th>PWC/kg W/kg</th>
<th>VO(_2) l/min</th>
<th>Recovery HR (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>&lt;21</td>
<td>15</td>
<td>73.3 ± 3.28</td>
<td>115.5 ± 3.11</td>
<td>73.3 ± 1.72</td>
<td>168.3 ± 7.5</td>
<td>173.7 ± 2.88</td>
<td>65.3 ± 2.69</td>
<td>200.2 ± 12.6</td>
<td>2.76 ± 0.17</td>
<td>3.59 ± 0.16</td>
<td>83.9 ± 3.28</td>
</tr>
<tr>
<td>2.</td>
<td>21-30</td>
<td>54</td>
<td>71.3 ± 1.64</td>
<td>125.5 ± 1.67</td>
<td>79.1 ± 1.11</td>
<td>173.6 ± 4.7</td>
<td>179.9 ± 2.9</td>
<td>71.8 ± 1.41</td>
<td>208.3 ± 7.3</td>
<td>2.66 ± 0.09</td>
<td>3.74 ± 0.08</td>
<td>80.8 ± 1.58</td>
</tr>
<tr>
<td>3.</td>
<td>31-40</td>
<td>42</td>
<td>73.9 ± 1.45</td>
<td>128.9 ± 2.02</td>
<td>84.7 ± 1.3</td>
<td>174.4 ± 6.9</td>
<td>183.8 ± 3.8</td>
<td>77.5 ± 1.7</td>
<td>212.9 ± 7.4</td>
<td>2.56 ± 0.08</td>
<td>3.84 ± 1.21</td>
<td>83.0 ± 1.36</td>
</tr>
<tr>
<td>4.</td>
<td>41-50</td>
<td>54</td>
<td>73.2 ± 1.46</td>
<td>127.3 ± 1.61</td>
<td>86.0 ± 1.08</td>
<td>168.5 ± 4.6</td>
<td>183.6 ± 3.1</td>
<td>85.3 ± 3.1</td>
<td>207.8 ± 6.1</td>
<td>2.5 ± 0.06</td>
<td>3.74 ± 0.08</td>
<td>82.3 ± 1.72</td>
</tr>
<tr>
<td>5.</td>
<td>51-60</td>
<td>29</td>
<td>74.8 ± 2.54</td>
<td>132.1 ± 3.26</td>
<td>88.0 ± 1.6</td>
<td>162.1 ± 6.4</td>
<td>190.8 ± 3.89</td>
<td>88.9 ± 2.22</td>
<td>203.0 ± 6.2</td>
<td>2.47 ± 0.08</td>
<td>3.67 ± 0.08</td>
<td>81.7 ± 2.5</td>
</tr>
<tr>
<td>6.</td>
<td>&gt;60</td>
<td>15</td>
<td>76.7 ± 2.85</td>
<td>132.7 ± 4.67</td>
<td>83.3 ± 2.84</td>
<td>123.3 ± 7.9</td>
<td>190.7 ± 5.56</td>
<td>80.3 ± 3.82</td>
<td>201.2 ± 9.4</td>
<td>2.63 ± 0.12</td>
<td>3.66 ± 0.12</td>
<td>78.5 ± 2.24</td>
</tr>
</tbody>
</table>
that the mean of the heart rate was the lowest at the age of 60 and over. At that age the heart rate's reaction to load is the smallest. No significant changes in heart rates at the recovery period according to age were found.

We found a moderate correlation between age and blood pressure (r=0.41). The correlation between age and blood pressure grew higher during the test (r=0.48). There is a positive correlation between blood pressure and serum cholesterol. Changes in the lipid metabolism favour forming of hypertonic processes in the organism (Reaven et al. 1989; Bona et al., 1991).

The amount of hypertonic reactions to physical load increases considerably with age (Fig 4). This indicates early changes in the haemodynamic regulation, increase in the peripheral resistance of arterial vessels and the beginning of development of hypertonic processes. The percentage of Estonian men with hypertonic reactions was higher than that of Estonian women of the same age (Täll et al. 1994).

![Fig 4. Occurrence of Hypertonic Reaction to Physical Load.](image)

The means of the PWC\textsubscript{170} and Vo\textsubscript{2}\text{max} do not show statistically significant dynamics with age, but PWC\textsubscript{170}/kg and Vo\textsubscript{2}\text{max}/kg were lower in the older groups. More important are individual indices because individual training programs are prescribed according to individual parameters.

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Fig 5. The frequency of ischemic changes in ECG during the test.

The frequency of ischemic changes in ECG at rest is small and age dynamics is minimal. The frequency of ischemic changes in ECG increased significantly during the test, especially in men over 40 years old (Fig 5).

Conclusion

As a result of the investigation of 209 men we collected data about their work characteristics, physical activity and fitness, and we also determined their morphological and laboratory indices. The collected data should be used for carrying out preventive measures, prescribing the amounts of physical load and increasing the resistance of organism.

References


Kokkuvõte

Eesti meeste kehaline aktiivsus, töövõime ning tervise üldpilt on saanud meditsiiniliseks ja ka sotsiaalseks probleemiks. Mehed haigestuvad sagedamini südame-veresoonkonna haigustesse ja suremus nendesse haigustesse on kõrgem, kui Eesti naistel.

varieeruvust. Märkimisväärsest suureneb isheemiliste muutuste hulk EKG-s koormuse ajal,

Saadud andmed on aluseks praktiliste soovituste andmisel kehalise aktiivsuse, toitumise ning elustiili kohta Eesti meestel.
Children in Competitive Sports

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In many kinds of sports intensive physical training and participation in competitions start already in childhood. Playground activities are replaced by intensive stereotypical requirements to the growing organism, with possible influence on the physical and mental development of the child. The participation of children in top sports can be regarded as a biological experiment where the athlete has a risk to exceed the limits of his organism for load tolerance and suffer health damage. The task of sports medicine in this process is to find out the contraindications to active sports and to ensure the safety of children via continuous monitoring.

To-date there are no sufficient data that would allow to draw a line between the training loads which can be considered as physiological, and those which may constitute health hazards and cause pathological damage to a growing organism.

Considerable individual differences have been found between the height, weight, and functional capacities in children of the same age (Mafulli 1992a, Birrer, Levine 1987). Heterogenity is evident also when comparing their anthropometrical data and biomechanical functions of the musculo-skeleton system. The anatomical individualities and differences in the biomechanical functions of joints, particularly in children with higher body weight, cause a greater demand for energy and a predisposition for sport injuries and health damage. The body proportions of a child change markedly when he grows older, e.g. in a 4-year-old the head forms 23% of the total body mass whereas in a 14-year-old it is 8%. In children the muscle strength is nor always proportional to their body mass and it may vary considerably among children of the same age. Hence, a big body mass does not automatically mean a high physical capacity. During the period of fast growth a relative slowing down in the increase of the indicators of strength has been observed and the fastness of learning new skills becomes somewhat inhibited, too.

In case of submaximal physical loads, in comparison with adults, children spend more energy and their metabolic reserves are
smaller, which are the causes why children get tired more quickly (Birrer, Levine 1987). With advancing age the response of the heart rate to physical loads becomes less manifest.

Until the end of puberty the age-related maximal working capacities in children show an increase linear correlation to those of the body measurements. Girls reach their maximum aerobic working capacities earlier than do boys, but in all age groups these data for girls are lower, forming 70 - 90% of the respective averages for boys. As the age increases, also the individual differences in the gain of the maximal aerobic working capacity grow age-related maximal aerobic working capacity is usually highest in 11 - 12 year old children (Rowland 1990).

Analyses of children’s anaerobic working capacity have shown that during a maximal physical effort their blood lactate levels are lower than in adults, indicating that in children a smaller proportion of energy is released by anaerobic glucoysis (Fagard, Bielen, Amery 1991). That explains why the duration of a maximal anaerobic effort in children is considerably shorter than in adults.

In children physical training produces a rise in the functional reserves of the organism. The physiological effect of training is, in principle, similar to that in adults, but the wider individual differences in children have to be taken into account.

Strength training brings about a gain in muscle strength in all age groups but the increase in indicators of strength and in muscular hypertrophy is faster after the end of puberty (Ramsay, Blimkie, Smith, Garner, MacDougall, Sale 1990). A certain slow-down in the increase of strength has been noted when trainings are conducted during the period of fast growth. Besides muscular hypertrophy, a smaller amount of fatty tissue is characteristic for trained children.

It has been observed that actively training children, compared to non-training children, show a faster increase in aerobic working capacity and a higher level of maximum oxygen consumption (Sundberg, Eloveninio 1982). The effect of endurance training becomes evident more slowly before and during puberty than after that. In well-trained girls the levels of maximal oxygen consumption may even exceed the levels of untrained boys of the same age. The vagotonical type of regulation of blood circulation has been seen more often in trained children.

Opinions differ as to the harmful effect of physical training and competitive sports to the development of children. There are data in general statistics confirming that children suffer from sport injuries more often than do adults (Gallagher, Finison, Guyer, Goodenough
1984). Girls who participate in competitive sports already at an early age have been noted to have a later menarche and more menstrual cycle disorders that their non-training peers (Bonen, Keizer 1984).

Based on the studies in rowing, ice hockey, and bicycling, intensive physical training does not affect the height gain of 12 - 15 year old boys (Mafulli 1992a).

Parallel to that there have been data suggesting that intensive physical training may affect the normal growth of children. It is thought that moderate physical training may stimulate the growth of bones whereas excessive loads may damage the epiphyseal plates and lead to an earlier end of ossification (Schwab 1977, Larson, McMahon 1966). Some researchers state categorically that intensive physical training and competitive sports, particularly before puberty, inhibit both the physical and the mental development of the child (American Academy of Pediatrics 1982, Sayne 1975).

Considering the physiological changes in a child’s organism, it is evident that the extent and intensity of trainings should be individualized according to the biological age of the child. As a general pedagogical principle it is advised that before puberty the physical training should mainly focus on the general development of the young organism and only later would switch over to the specific training of one or another kind of sport (Mafulli, Pintore 1990).

When shaping the psycho-motor skills in children, wide individual differences are met, just as there are considerable differences between their abilities to realize their skills and potential at competitions (Ogilvie 1979).

As to the factors of the outer environment, the child organism is not as adaptive as an adult’s: it is less tolerant of high temperatures, middle and high mountain conditions, etc. Health damage can easily develop in such conditions unless the intensity and extent of physical trainings is corrected (Sport Medicine Manual 1990).

It has been advised that when planning the general training loads, the rule of 10% should be followed: the loads for children should always be at least 10% smaller than those for adults (Mafulli 1992a).

It is particularly important that during the process of trainings and competitions the children’s state of health and their dynamics of physical working capacity should be continuously monitored through regular medical examinations and medical-pedagogical observations and tests. The children themselves, too, have to be taught certain methods that enable them, for self-control, to discover signs of deleterious effects of intensive physical training. Continuous monitoring of the heart rate at
the trainings (by means of "Sporttester") allows one to choose the right intensity so that it would exactly correspond to the actual physical capacity of the child's organism.

Successful and unharmed-for-health participation of children in competitive sports depends much on the cooperation between the coach, the physician, and the parents. It is essential for all the parties to have the correct orientation and the self-critical attitude that ensure that the health and the normal development of children have the priority over forcing the pace to high achievements in sports.

References.


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Kokkuvõte

Paljudel spordialadel alustatakse intensiivseid kehalisi treeninguürit ja osalemist võistlusspordis juba varases lapseeas. Spordimeditsiini ülesandeks selles protsessis on tagada laste normaalne vaimne ja füüsiline areng ning tervisekahjustuste vältimine. Ülevaates käsitletakse erinevate kehaliste koormuste füsioloogilist mõju lapse organismile ja analüüsitakse üldisi põhimõtteid treeningkoormuste planeerimiseks ealisest aspektist. Sportlikud eesmärgid ei tohi kahjustada lapse tervist ja normaalset arengut. Selleks on vaja õigel orientatsiooni ja kriitilist hoiakut nii treenerite, arstide kui ka lapsevanemate poolt.
MYOCARDIAL PERFUSION IMAGING AND CORONARY ANGIOGRAPHY IN THE DIAGNOSTICS OF CORONARY ARTERY DISEASE

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Abstract — Assessment of myocardial perfusion during exercise is an important factor in the detection of ischaemic heart disease. We studied 23 patients (21 males, 2 females) with coronary artery disease by means of veloergometric stress test. 99Technetium-MIBI was injected intravenously: 185 MBq at the highest achievable level of exercise, and 740 MBq at rest. After both injections imaging in 3 projections was performed. The studies were performed by planar gamma camera Pho Gamma IV. Besides 10 patients underwent coronary angiography. The mean age of the patients was 52 ± 9 years. The mean percentage of the age-related peak heart rate achieved during exercise was 85%, with an absolute value of 138 ± 23 beats per minute. In all the patients changes of ST-segment were observed; 12 had normal myocardial perfusion, and 9 showed persistent defects in myocardial images which in 7 cases were combined with ischaemia induced by the exercise. Two patients demonstrated transient perfusion defects. A post-test examination for coronary angiography was performed in 43%. From the patients who underwent coronary catheterization, 2 had persistent defects in their myocardial images. These 2 patients also had a significant (>75%) reduction of the coronary artery lumen as detected by coronary angiography. In 8 patients myocardial perfusion was found to be normal and they showed angiographically a reduction below 75% of the luminal diameter. In conclusion, our results demonstrated that nuclear myocardial perfusion imaging is a reliable method for the identification of myocardial necrosis and ischaemia in coronary artery disease patients with a critical coronary artery stenosis.

Key words — coronary angiography, myocardial perfusion, nuclear imaging
Introduction

Assessment of myocardial perfusion during exercise is an important factor in the detection of coronary artery disease (CAD). Imaging techniques determine myocardial perfusion and cellular integrity. The left ventricular function and myocardial perfusion are important prognostic indicators for patients with CAD, and information on these two can be valuable in the planning of individual patient management. Myocardial perfusion scintigraphy with $^{99}$Technetium-MIBI ($^{99}$Tc-MIBI) can determine the presence, localization, and extent of ischaemic regions (Jones 1984, Gerundi 1986, Okada 1988, Glover 1990, DiRocco 1992, Frey 1992). This technique is increasingly employed as a noninvasive method (Beller 1993, Ciavolla 1993).

The aim of the present study was to compare the results of myocardial perfusion exercise study with those of coronary angiography (CAG) in CAD patients with diagnostic problems.

Subjects and Methods

Twenty-three patients (21 males, 2 females) were referred to the Department of Radiology of Tartu University Hospital for exercise $^{99}$Tc-MIBI testing for the detection or evaluation of CAD. Nine patients had had a previous myocardial infarction. The mean age of the patients was 52±9 years. Veloergometric stress test was performed with an initial external load of 50 W for 3 min for males, and 25 W for 3 min for females. Thereafter the load was increased every 3 min by 25 W until one of the following criteria was fulfilled: severe angina pectoris, an ischaemic ST-segment depression of at least 0.2 mV, dyspnea, manifest hypoxia or hypertension, or the patient reached the age-predicted maximal heart rate. In all patients, medicaments such as beta-blocking agents and calcium antagonists were discontinued 24 hours before the test. At the highest achievable level of exercise, 185 MBq $^{99}$Tc-MIBI was injected intravenously and exercise was continued for one more minute. Imaging was started 30-60 min after the cessation of exercise in 3 projections: in the anterior, 45° and 70° left anterior oblique. Four hours later, at rest, 740 MBq $^{99}$Tc-MIBI was injected intravenously and imaging was performed in the same projections. The condition of the myocardial walls was evaluated in 3 different projections, with 5 regions of special interest: anterior (AN), septal (S), apical (AP), inferior (I), and lateral (L). The studies were performed by planar gamma camera Pho Gamma
IV (Searle). Each view was obtained during 8 -10 min, with a minimum of 400 000 counts in the field of view.

Results

The characteristics of myocardial perfusion images, clinical and hemodynamic results of the $^{99}$Tc-MIBI exercise test are given in Table 1.

Table 1. Individual characteristics of myocardial perfusion images, clinical and hemodynamic results

<table>
<thead>
<tr>
<th>Pat no</th>
<th>Age</th>
<th>Prev MI</th>
<th>EHR bpm</th>
<th>ESBP mmHg</th>
<th>PM</th>
<th>ST changes (depression)</th>
<th>$^{99}$Tc defect</th>
<th>Symptoms</th>
</tr>
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<tr>
<td>1</td>
<td>45</td>
<td>Y</td>
<td>132</td>
<td>130</td>
<td>80</td>
<td>II,III,aVF,v2-v6</td>
<td>AN,S,I(P+T)</td>
<td>angina</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>N</td>
<td>135</td>
<td>135</td>
<td>80</td>
<td>v3-v6</td>
<td>AN(P+T)</td>
<td>BP↓</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>N</td>
<td>168</td>
<td>210</td>
<td>109</td>
<td>v2-v6</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>N</td>
<td>140</td>
<td>155</td>
<td>81</td>
<td>v2-v6*</td>
<td>AN,S,AP(P+T)</td>
<td>angina</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>N</td>
<td>110</td>
<td>200</td>
<td>68</td>
<td>I,II,aVL,v2-v6</td>
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<td>angina</td>
</tr>
<tr>
<td>6</td>
<td>54</td>
<td>N</td>
<td>158</td>
<td>210</td>
<td>100</td>
<td>v4-v6</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>45</td>
<td>N</td>
<td>150</td>
<td>170</td>
<td>85</td>
<td>I,II,aVL,v2-v6</td>
<td>ST↓</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>61</td>
<td>N</td>
<td>154</td>
<td>210</td>
<td>100</td>
<td>I,II,aVL,v3-v6</td>
<td>dyspnea</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>54</td>
<td>Y</td>
<td>100</td>
<td>160</td>
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<td>angina</td>
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<td>255</td>
<td>88</td>
<td>v4-v6</td>
<td>BP↑</td>
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<tr>
<td>12</td>
<td>67</td>
<td>N</td>
<td>125</td>
<td>170</td>
<td>61</td>
<td>v3-v6</td>
<td>angina</td>
<td></td>
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<td>13</td>
<td>52</td>
<td>Y</td>
<td>125</td>
<td>185</td>
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<tr>
<td>14</td>
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<td>190</td>
<td>140</td>
<td>118</td>
<td>v2-v6</td>
<td>AN,I(P+T)</td>
<td>100%</td>
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<tr>
<td>15</td>
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<td>Y</td>
<td>115</td>
<td>145</td>
<td>77</td>
<td>v4-v6</td>
<td>I,AP(T)</td>
<td>dyspnea</td>
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<tr>
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<td>155</td>
<td>150</td>
<td>95</td>
<td>II,III,aVF,v2-v4*</td>
<td>AN,L(T)</td>
<td>100%</td>
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<td>100</td>
<td>II,III,aVF,v2-v6</td>
<td>I(P)</td>
<td>100%</td>
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<tr>
<td>18</td>
<td>52</td>
<td>N</td>
<td>140</td>
<td>195</td>
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<td>dyspnea</td>
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<td>60</td>
<td>Y</td>
<td>125</td>
<td>195</td>
<td>83</td>
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<td>I(P+T)</td>
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<td>38</td>
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<td>v5-v6</td>
<td>dyspnea</td>
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<td>23</td>
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<td>N</td>
<td>115</td>
<td>190</td>
<td>71</td>
<td>I,II,v4-v6</td>
<td>angina</td>
<td></td>
</tr>
</tbody>
</table>

Prev MI=previous myocardial infarction; EHR=exercise heart rate; ESBP=exercise systolic blood pressure; PM=predicted maximum; *=elevation of ST
In all patients changes of the ST-segment during exercise were observed: in 19 patients depression, and in 4 patients elevation. The mean percentage of the age-related peak heart rate achieved during exercise was 85%, absolute value 138±32 beats per minute (bpm). The exercise test was terminated in 9 patients due to severe angina, in 4 due to dyspnea, in 2 due to hypertension, in 1 patient due to hypotension, and in 2 patients due to severe ischaemic ST-segment depression. Five patients reached the age-predicted maximal heart rate. In 12 patients the myocardial perfusion was found to be normal. Nine patients had persistent (P) defects in myocardial images which in 7 cases were combined with ischaemia induced by the exercise. Two patients demonstrated transient (T) perfusion defects. Both anterior and inferior defects were detected in 7 subjects, septal in 4, apical in 3, and lateral in 2 cases. Besides, 10 patients underwent CAG. The comparison of the perfusion images and the CAG findings is presented in Table 2.

Table 2. Comparison of results of myocardial perfusion scintigraphy and coronary angiography

<table>
<thead>
<tr>
<th>Patient no</th>
<th>A</th>
<th>C</th>
<th>OM</th>
<th>D</th>
<th>Di₁</th>
<th>99Tc defect</th>
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<tr>
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<td>2</td>
<td>5</td>
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<td>3</td>
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<td>AN, AP, S</td>
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<td>3</td>
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<td>2</td>
<td>3</td>
<td>2</td>
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</tbody>
</table>

A=ramus interventricularis anterior; C=ramus circumflexus; OM=ramus obtusus marginalis; D=arteria coronaria dextra; Di₁=ramus diagonalis
Stenosis by CAG: 1=25%; 2=26-50%; 3=51-75%; 4=76-90%; 5=91-99%; 6=100%

From the patients who underwent CAG, 2 had persistent defects in their myocardial images, and 1 of them a defect combined with ischaemia induced by the exercise test. These 2 patients also had a significant reduction (over 75%) of the coronary artery lumen as detected by CAG.
Eight of the patients who underwent CAG had a normal myocardial perfusion. All these patients had angiographically a reduction below 75% of the luminal diameter.

Discussion

The diagnosis of CAD is a difficult problem in some patients. Previous data in the literature have demonstrated the utility of $^{99m}$Tc-MIBI for identifying and localizing significant coronary stenosis (Gigson 1992, Taillefer 1992, Verzijlbergen 1992). Nuclear exercise tests are often used by radiologists as a noninvasive relatively inexpensive technique which makes them preferable to CAG. It has been shown that nuclear tests significantly reduce the need for coronary catheterization among CAD patients (Maublant 1988). For a more precise diagnosis, we performed, in addition to the nuclear exercise test, also CAG in 43% of the patients (in 10 cases of 23). All these patients were symptomatic during the exercise test but only 2 of them had significant defects in their myocardial perfusion images. These 2 were the only ones who had a critical stenosis of the lumen as detected by CAG.

In conclusion, our results demonstrated that nuclear myocardial perfusion imaging is a reliable method for the identification of myocardial ischaemia and necrosis in CAD patients with critical coronary artery stenosis.

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Ventricular Wall Motion Studies Evaluated From Gated Myocardial Perfusion Studies. Am Heart J 123: 59-68

Kokkuvõte


Meie uuringud näitasid, et südamelihase perfusiooni isotoopuuring on informatiivne meetod koronaarverevarustuse häirete ja müokardi nekroosi kindlakstegemiseks.
Abstract — Electrical impedance methods used in practice for stroke volume estimation, though having advantageous features, have their weaknesses, too. In the paper a survey of the methods is given with respect to improvement them using computer simulation and advanced signal processing. As well the Kubicek and Sramek methods as the Tischenko method are shown to be considerably empirical and unsuitable for such kind of modernisation. Improvement of electrical impedance methods for stroke volume estimation would be realised by using new technique - electrical impedance tomography.

Key words — impedance plethysmography, impedance cardiography, impedance tomography, bioimpedance, integral rheography, stroke volume, cardiac output, Kubicek method, Sramek method, Tischenko method

Introduction

Electrical impedance methods are based on measurement of the electrical impedance (i.e., the modulus of the full complex resistance) of the human body or its parts at sub-high frequency (20...200 kHz). It has been found that the impedance of body pulses in synchronism with the heart. This pulsation is the origin of impedance plethysmogram. Amplitude of the impedance plethysmogram comprises about 1/1000 ... 1/100 of the mean value of the observed impedance. Such a phenomenon is explained as a result of an impact of pulsation of blood content in tissues on the electrical resistivity of the tissues (Nyboer et al. 1950). Besides, considerable effect of changing the resistivity of blood itself with its linear velocity is detected (Moskalenko et al. 1959, Liebman et

This work was supported by the Estonian Science Foundation grant No. 272.
al. 1968, Dellimore et al. 1975), too. Several methods have been developed on the basis of described phenomenon for estimation of the volume flow of blood in different organs or parts of the body.

The beginning of use of the electrical impedance methods for the observation of physiological phenomena was in the second half of the 1930ies in Germany. Afterwards these ideas have spread to Austria, Russia, USA etc. The first actual model of genesis of the impedance plethysmogram was proposed by Jan Nyboer (Nyboer et al. 1950). He has derived a formula for calculation of the increment of blood volume $\Delta V$ from the corresponding decrement of electrical impedance $\Delta Z$ for the case of a cylindrical region of body (like arms or feet) that we can write as follows:

$$\Delta V = -\frac{\rho L^2}{Z^2} \Delta Z .$$

(1)

Here $\rho$ is the resistivity (more exactly, the specific impedance) of blood, $L$ is the of the cylindrical section of body included into the measurement circuit (equated to the distance between the measurement electrodes). $Z$ is the mean value of the measured impedance.

(1) assumes the so-called parallel conductor model (Shimazu et al. 1982, Patterson 1989) of the impedance plethysmogram genesis: 1) the impedance change is assumed to be caused only by delivery of an additional amount of blood to the measurement region, 2) the additional blood volume in its turn is assumed to form a cylindrical region that electrically is connected in parallel with the main one.

As described above, $\Delta V$ and $\Delta Z$ vary with time quasi-periodically, $\Delta V = \Delta V(t)$, $\Delta Z = \Delta Z(t)$, having the frequency of the heart. $Z$ drifts slowly and, besides, changes typically in synchronism with breathing, $Z = \bar{Z} (t)$. Thus Nyboer has introduced splitting the time course of the measured impedance into two additive components

$$Z(t) = \bar{Z}(t) + \Delta Z(t) .$$

(2)

having different magnitudes and different spectra.

Later W.G. Kubicek and co-authors used the basis founded by Nyboer to derive their method for estimation of the stroke volume (SV), i.e. volume of blood, put out by a ventricle of the heart during a contraction cycle. In the USSR M.I. Tischenko put forward his own method to do it, following the Russian tradition of impedance plethysmography (called rheography). Whilst having some non-principal modifications, these two methods remain as the main electrical impedance methods for
estimation of stroke volume (and cardiac output, close connected with it) up to nowadays. In similar manner

a

b

Figure 1. Arrangement of electrodes at the recording of the electrical impedance for SV estimation: a - the Kubicek impedance cardiography, b - the Sramek bioimpedance method, c - the Tischenko integral rheography.

c

various methods for measurement of local (organ) blood flows were proposed.

The Kubicek method

This is an electrical impedance method for the estimation of the stroke volume. It was developed in the middle of 1960ies by a group, leader of which is considered to be William G. Kubicek, and was named impedance cardiography (Kubicek et al. 1966, 1970; see Patterson 1989). The Kubicek method constituted a new quality at its time of come out: a new technical solution and an advanced (relative to that time) signal processing algorithm were introduced.

Up to that time bridge circuits were used for impedance measurement in the impedance plethysmography. Kubicek’s co-author Robert Patterson developed the impedance cardiograph where four-
**electrode technique** (Fig. 1.a) was used. This technique would enable to minimise the noise caused by variation of electrode-to-skin contact resistance due to movements of patient. Special band electrodes put around the body were introduced. As the output impedance of the current source and the input impedance of the voltage amplifier can be very high, the changes of the contact resistance would affect not to the measurement current nor to the measured voltage.

The band electrodes later have came under criticism (Sramek 1986). Indeed, using of the band electrodes could replace the old mechanism of noise generation with another. Namely, in case of a non-uniform distribution of the contact resistance between the band and the body, the area of the best contact (the effective centre of location of the electrode) can drift on the surface of the body under the band. This will cause noise, because the effective location of the current leading electrodes or the position of the potential electrodes relative to the equipotentials will change. (This defect would be eliminated, if we divided the bands into several less electrodes and connected each of them with the lead cable by proper resistors.)

In case of perfect contact between the body and the band electrode, the band would enable to unify the distribution of potentials on the surface of body. Thus in this ideal case it makes for use the cylindrical conductor model of the body. But the calculation formula of the Kubicek method is just based on the cylindrical conductor model: the Nyboer's formula (1) has been boldly applied to such a complicated region as the thorax. This approach will be discussed further.

A typical Kubicek impedance cardiogram $\Delta Z = \Delta Z(t)$ of a normal healthy man is shown on Fig. 2, curve a. Here traditional inverse depiction is used where the direction of $Z$ increase is downward. Fig. 2, curve b represents its time derivative $dZ(t)/dt$, in inverse depiction too. Traditionally (Lababidi et al., 1970) its characteristic points are labelled as shown here.

**Figure 2.** The Kubicek impedance cardiogram of a normal healthy man (curve a) and its time derivative (curve b). Curve c represents ECG R-wave timing pulses.
The ideology of the Kubicek method includes the so-called Kubicek's (it ought to be called Patterson's) extrapolation. It tries to take account of outflow of blood from the measurement region (that takes place in the same time as the cardiac ejection occurs). The original justification of the Kubicek's extrapolation by its authors (Kubicek et al. 1966; Patterson 1989) was poor. The extrapolation consists an expedient to calculate, on the basis of the measured signal, the magnitude of the full impedance decrement $\Delta Z_{sv}$, that ought to correspond to the stroke volume:

$$\Delta Z_{sv} = \min_{teCycle} \frac{dZ(t)}{dt} \cdot T_E . \quad (3)$$

Here $T_E$ is duration of heart ejection (that can be estimated using as well impedance as non-impedance methods, for instance phonocardiography), $dZ(t)/dt$ is time derivative of the registered impedance, formula (3) contains its minimum (peak) value for the heart cycle in operation. Putting into (1) the impedance decrement from (3) one gets the Kubicek formula for the calculation of stroke volume $\Delta V_{sv}$ (Kubicek et al. 1970):

$$\Delta V_{sv} = -\rho \frac{L^2}{Z^2} \cdot \min_{teCycle} \frac{dZ(t)}{dt} \cdot T_E . \quad (4)$$

In practice impedance cardiograms often are registered at breathing, therefore breathing component is present in records then. Differentiation suppresses the lower frequencies of spectrum, thus the breathing component is significantly suppressed in $dZ(t)/dt$ signal. That is why in case of the Kubicek method breathing influence has much less effect than in any method that uses the amplitude of the impedance plethysmogram itself.

The Kubicek's extrapolation is one of the weakest elements of the ideology of the Kubicek method. The work by Rubal and co-authors (Rubal et al., 1980) suggests that as the second multiplier of the product on the right side of (3) is the duration of the heart ejection period, therefore the success of the Kubicek's extrapolation must be caused by existence of a correlation between the peak value of $dZ(t)/dt$ and the maximum flows in the aorta and, perhaps, in the pulmonary artery. Furthermore, it has been speculated (Sramek 1986, Ovsyshcher et al. 1993) that the wave form of the time derivative of the Kubicek impedance cardiogram is to certain degree similar to the flow wave forms in the named arterial vessels. The Kubicek's extrapolation seems to have its
success due to this fortunate, however irregular and uncomplete, similarity.

Absolute calibration of the Kubicek impedance cardiography is a widely discussed aspect of the method. In their first publications (Kubicek et al. 1966) the authors of the method suggested a constant empirical calibration coefficient of value 0.9 in the calculation formula. Further the location of the lower potential electrode was shifted 2 cm upward and was canonised at the level of xiphoid joint (Kubicek et al. 1970). Thus the calibration coefficient was omitted in the calculation formula. Boer and colleagues have shown (Boer et al. 1979) that the value of SV calculated according to Kubicek highly depends (for the lower electrode about +4.5% per cm) on the distance between the potential electrodes if they are shifted away from the canonical locations, even in case of the same patient at the same state. Thus we can conclude that the absolute calibration of the Kubicek method is empirical and the calculation formula does not evidently contain any calibration coefficient only due to suitable choice of the location of the lower potential electrode. Boer's result shows, besides, that one will easily get erroneous absolute values of SV if one does not accurately locate the potential electrodes. If the Kubicek formula (i.e., the cylindrical parallel conductor model) was more valid then such a dependence on the distance between the electrodes would not appear. It would be natural for the cylindrical conductor that the characteristics of the measured impedance and the distance between the electrodes changed in accordance with each other so that one could get the same SV estimat, independent of the deviations of the distance between the potential electrodes.

More than 20 years after designing the Kubicek impedance cardiography Robert Patterson published (Patterson 1989) his opinion formed about the method. Patterson presented data recorded in animal experiments. These data suggest that the Kubicek impedance cardiogram is determined as well by hemodynamic processes in systemic circulation as by processes in pulmonary circulation. (This fact has been clearly demonstrated yet before (Geddes et al. 1972).) Furthermore, the contributions of the both vary with individual anatomical and physiological characteristics. Naturally the heart has its contribution, too. Thus it can be concluded that the Kubicek impedance cardiogram constitutes only an integral view to a complex of complicated processes in a complicated spatial area, whereby a considerable number of characteristics or parameters describing the contributions of different processes are individually determined.
As the SV value is of great clinical importance and there is scarcely any other non-invasive method for its continuous measurement, a great number of clinical evaluations of the Kubicek impedance cardiography have been carried out (see Ovsyshcher et al. 1993). Started by the authors of the Kubicek method, comparisons of the method with other (mostly invasive) methods were carried out and various contingents were studied.

A quite wide range of correlation coefficient $r$ values for the impedance cardiography and the comparative methods can be obtained in publications: $r = 0.4...0.99$. In typical evaluations $r$ was found to be $0.7...0.94$.

The evaluations suggest that the method is plausible in case of normal healthy adults. But there are shortcomings in critically ill ventilated patients, in pregnant women, and difficulties or dissonances in children with intracardiac shunt, in adults with valvular regurgitation and low cardiac output. Thus just the patients who most need careful watching for cardiac output cannot be reliably treated with the impedance cardiography method. Comparing Fig. 3 to Fig. 2 one can see an example of differences between a patient's and a healthy man's impedance cardiograms that can cause difficulties.

![Figure 3. The Kubicek impedance cardiogram of a patient, having mitral and aortic valve prostheses in combination with pacemaker, (curve a) and its time derivative (curve b). Curve c represents ECG R-wave timing pulses. Label pm indicates pacemaker artifacts.](image)

Besides, one must notice that the evaluations have been carried out using correlation (i.e., statistical) methods. This fact reinforces opinion that there is no individual measurement of the value of SV with the impedance cardiography method yet but only statistically fitted estimation. It is generally considered that the existing impedance cardiography can rather serve for continuous watching the relative
dynamism of SV than for sparse or single measurements of its absolute value.

To improve the diagnostics and watching for the cardiovascular patients who have their individual (unknown in advance) peculiarities, physicians would have something more reliable.

**Modifications of the Kubicek method**

**The Sramek method**

As the Kubicek method has certain shortcomings, several attempts to improve it were undertaken. Most of such modifications have little popularity. There are attempts to better account the individual dimensions of body (Storozhenko et al. 1983) caused by experience indicating such kind of lack in the Kubicek method. Location of the electrodes has undergone a non-principal modification (Pushkar' et al. 1977): the upper current electrode has been recommended to put around the head. As (4) includes blood resistivity $\rho$ as a scaling factor, methods are introduced to count its value from the individual blood test data (Mohapatra et al. 1977).

Since the Nyboer's formula (1) is significantly simpler if expressed not for the impedance $Z$ but for its reciprocal value $Y = 1/Z$ (named admittance), a modification named admittance plethysmography was introduced (Ito et al. 1976, Shimazu et al. 1982). If admittance is measured then the mean value of it will not be needed for SV calculation. But the equipment for this modification is technically more complicated.

There are several versions of the impedance derivative peak value measurement. The authors of the Kubicek method suggested to measure the C-wave (Fig. 2) amplitude from the zero line of the derivative. Lababidi and co-authors have recommended (Lababidi et al. 1970) to measure it from the B point level of impedance cardiogram. As this can be difficult because the B point is hardly detectable there is a version of measurement it from the X point level, with posterior appropriate calibration (Zhang et al. 1986).

**The Sramek method** constitutes an essential modification of the Kubicek method. It was proposed by B. Bo Sramek and co-authors in 1980ies (Sramek 1986) and it is called bioimpedance technology by them. There were reasons why this method has been proposed and has gained acceptance. The first reason is the described above lack of band electrodes which causes unexpected noise. To eliminate it, spot elec-
rodes (like used for ECG) were introduced. They are put in 8 points on the lines where the band electrodes were laid, in convenient places on the both sides (Fig. 1.b).

The second reason of modification was difficulty with the resistivity (specific impedance) of blood. It is quite unhandy to measure the resistivity of patient's blood (or haematocrit, for regressive calculation of resistivity). Sramek and co-authors have succeeded to avoid this need.

By splitting the square of basis impedance in (4) into product $Z^2 = \bar{Z} \cdot \bar{Z} = \rho L/S \cdot \bar{Z}$, they got a volume-dimensional coefficient in the formula. It was declared to be an individual parameter (i.e., calibration coefficient) and was named \textit{volume of electrical participating tissue} $V_{EPT}$. Using statistical and experimental data, a regression has been estimated:

$$V_{EPT} = \frac{P}{P_{IDL}(H)} \cdot \frac{(0.17H)^3}{4.2} \quad (5)$$

Here $P$ is the actual weight of the patient, $P_{IDL}(H)$ is its ideal value ("ideal weight"), that is a function of patient's height $H$. Function $P_{IDL}(H)$ is different for men and women, it must be corrected on the constitutional type of the patient.

The formula for the calculation of stroke volume $\Delta V_{SV}$ in case of the Sramek method looks following:

$$\Delta V_{SV} = -\frac{V_{EPT}}{Z} \cdot \min_{\text{Cycle}} \frac{dZ(t)}{dt} \cdot T_E \quad (6)$$

Though the method has useful advantages (does not need determination of $\rho, L$), it seems to be a step towards the fully-regressive estimation of SV on the basis of only anthropometrical data. Thus we could get only mean statistical estimates but not individual measured values for the concrete person with his/her possible pathology.

As the formula (6) contains only the first power of $\bar{Z}$, it may have dependence on the misplacement of electrodes that is different (and possibly more favourable) from the Kubicek's one.

About the spot electrodes R. Patterson has noted (Patterson 1989): "Past work attempting to use spot electrodes on thorax to measure directly ventricular volume produced a much variable signal [than in case of the Kubicek method, J.V.] and, therefore,
consistent quantitative results could not be obtained. Reproducibility continues to be one of the outstanding features of the band-electrode arrangement."

Yet the modifications introduced by Sramek do not change the general character of impedance cardiography. In this version the impedance cardiography even more constitutes a half-empirical method that enables only statistically calibrated SV estimation.

The Tischenko method

The method was developed by M.I. Tischenko in the beginning of 1970ies (Tischenko 1973, Tischenko et al. 1973). By the author it was named integral rheography. This designation refers to fact that this method comprises the whole human body as the object of measurement. A pair of connected together electrodes is put onto the limbs around the ankles and another similar pair is put onto the arms around the wrists (Fig. 1.c). The impedance between the wrists and the ankles is measured. The author of the method considered important some technical details that we now may conceive as non-principal. For instance, the original method prescribes using a bridge circuit, the measurement frequency has to be equal to 30 kHz.

The integral rheogram slightly differs from the Kubicek impedance cardiogram (compare Fig. 4 to Fig. 2). But the general forms of the both are quite similar. Therefore one can conclude that the main mechanism of genesis ought to be the same in the both cases. Tischenko himself considered his method to be an integral view of processes in the large blood vessels. We should agree with him.

![Figure 4. The Tischenko integral rheogram (curve a) of a normal healthy man (the same person as on Fig. 2) and its time derivative (curve b). Curve c represents ECG R-wave timing pulses.](image)
The Tischenko method offers formulae for calculation several physiological indices of man. For instance, the amount of extra-cellular liquid can be calculated. But the main aim of the method is to enable the SV estimation.

The SV calculation ideology is based on the Nyboer's formula (1) again. This time it is applied to the longitudinal tubes in the human body: the large blood vessels - the aorta and arteries. In distinction to the Kubicek method, no sophisticated expedient to account the venous outflow is taken. The amplitude of the impedance pulse serves as the indicator of SV. It must only have an appropriate calibration. If the designations introduced above are used, the initial theoretical formula for SV calculation according to Tischenko will look as follows:

\[ \Delta V_{SV} = -\frac{T_c}{KT_D} \cdot \frac{\rho L^2}{Z^2} \Delta Z_{MAX} \]  

Here \( \Delta V_{SV} \) means the stroke volume, \( L \) is the distance between the foot and the arm electrodes measured along the main arteries, \( T_c \) is the duration of heart cycle, \( T_D \) is the duration of the catacrotic ("falling", if the traditional inverse depiction is used) part of impedance curve, \( \Delta Z_{MAX} \) is the maximal impedance decrement, \( Z \) is the mean value of impedance, \( \rho = 150 \ \Omega \cdot \text{cm} \) is fixed value of blood resistivity, \( K \) is an empirical calibration coefficient.

The initial formula has been transmuted further. Though \( K \) varied in experiment in quite wide range (\( K = 0.27 \ldots 0.44 \)), the author of the method has found a strong correlation (\( r = 0.985 \)) between \( K \) and \( Z \), such that \( K \cdot \bar{Z} = 100 \ \Omega \). So the power of \( \bar{Z} \) has been reduced and a newly combined calibration coefficient has been introduced. Another transmutation has been done due to the length \( L \) of the longitudinal arterial tubes that had to be accounted. As this length correlated with the height \( H \) of person, corresponding regression has been built into the calculation formula. The regression obtained for men differed from the one got for women and therefore the resulting practical formula can be presented as follows:

\[ \Delta V_{sv} = k \cdot \frac{H^2}{\bar{Z}} \cdot \frac{T_c}{T_D} \cdot \frac{\Delta \zeta_{MAX}}{d\zeta/dZ} \]  

where \( k = 2.75 \ \text{cm} \) for men and \( k = 2.47 \ \text{cm} \) for women. Following the original publication (Tischenko 1973), the formula (8) is given here
in a form proper for manual handling of plotted records. Thus $\zeta$ is deviation of the plot of impedance from its base line and $\Delta \zeta_{\text{MAX}}$ is the amplitude of the rheogram (measured in cm, for instance), $d\zeta/dZ$ is the sensitivity of recording system (correspondingly in cm/Ω). By the author the sensitivity was prescribed to estimate for every measurement, turning on a special calibrating resistor.

The explicit use of calibration here seems to be caused by the bridge measurement technique. The apparatus may be unstable and bridge would change its sensitivity with changing the individual mean impedance of body. However, the method can be realised using the four-electrode technique as well.

One can notice a similarity between the formulae (6) and (8) in the next sense: 1) unlike the Kubicek formula (4), $Z$ is in the first power in the both, 2) the distance between the main electrodes is reduced (using statistical regression) to some usually measured anthropometrical data. The priority of this art evidently belongs to Tischenko.

All the foregoing demonstrates that the Tischenko method has qualitatively the same level of physical and physiological justification and is as well empirical and statistical as the Kubicek's and Sramek's ones.

As for comparative studies of the Tischenko method with other methods, there are quite few investigations. For instance, the author of the method has been compared it to the direct Fick method (resultant correlation coefficient was $r = 0.99$, $n = 28$ patients have been involved), to acetylene rebreathing method ($r = 0.84$, $n = 31$, healthy people) and to thermodilution method ($r = 0.95$, $n = 25$, patients) (Tischenko et al. 1973). The correlations look very good but there are too few investigations. While there are hundreds of investigations of the Kubicek method, the number of investigations of the Tischenko method seems to be several tens times less. Hence the weak points of the Tischenko method can be not found yet.
Perspective

We saw above that the practical electrical impedance methods for SV measurement are still staying as half-empirical. The model of electrical conduction used is the Nyboer's parallel conductor model for a cylindrical region. Only rough depiction of haemodynamics is assumed. The modifications introduced have not changed this general character but only some details.

From the viewpoint of a biomedical engineer a following task could be set: to develop a model that with sufficient precision described the genesis of signal in case of any impedance plethysmographic method. This model must include co-operating model of haemodynamics and model of electrical conduction of the watched part of human body. If building of such a model for any existing method of SV estimation is impossible or too difficult, a new method of measurement must be developed. Then with the help of the model it would be possible to improve the methods for SV or blood flow estimation. A preliminary attempt of such approach exists already (Girling et al. 1979).

Simulation of the human circulation has intensively developed during last decades. Successful models of arterial haemodynamics have been built (Burattini et al. 1989, Campbell et al. 1990). There are hopeful attempts of simulation of overall circulatory dynamics in man, including arterial, cardiac and pulmonary dynamics of blood (Hardy et al. 1982, Vedru 1988). (However, venous haemodynamics has still remained a difficult to simulate subject because of its irregularity and individuality.)

Several attempts to develop conduction models for the impedance plethysmography have been done, mainly in connection with the Kubicek method. Simulation investigations of the distribution of electrical current in human thorax were carried out. Knowing the current distribution one can find contributions of different parts of the thorax and internal organs to the impedance plethysmogram.

Let us suppose the very complicated resistivity $\rho$ or conductivity $\sigma = 1/\rho$ distribution in the human body to be known. As the current density in a volume conductor is determined by the local value of the conductivity and the distribution of the electrical potential $\varphi$, the problem above will be solved by finding the potential distribution. This problem, the direct problem of electrical impedance plethysmography, is well-known and has been solved by physicists in principle. The un-
known potential $\varphi$ must satisfy the following partial differential equation

$$\sigma \Delta \varphi + \nabla \sigma \cdot \nabla \varphi = 0$$

(9)

under the Neumann-type boundary condition

$$\sigma \frac{\partial \varphi}{\partial n} = j$$

(10)

on the surface of the body. Here $j$ is the density of external electric current that is led to the surface of the conductive region ($j \neq 0$ only under the electrodes). The conductive object (i.e., human body) constitutes a closed 3-dimensional region having complicated distribution of conductivity $\sigma$. Using finite elements or difference methods has enabled to solve numerically the direct problem for suitably simplified models (Patterson 1985, Kosicki et al. 1986, Kim et al. 1988).

Such investigations have shown that almost all the regions of the thorax do have considerable contribution to the Kubicek impedance cardiogram. Besides, the contribution of the blood movement to impedance cardiogram has been investigated by simulation. It occurred (Kosicki et al. 1986, Kim et al. 1988) to have about same magnitude as the contributions of dilatation of large blood vessels, lung and heart volume changes.

This result accords with the experimental findings referred above. On these grounds conclusion can be done that at least in case of the Kubicek (or Sramek) method for impedance measurement, attempt to build a more adequate electrical conduction model than the Nyboer's one has little chance of success. Such a model has to make allowances to too many individually varying (i.e., unknown) parameters that characterise the human thorax as a volume conductor and as a part of circulatory system as well. Therefore it could not help to improve the SV estimation.

It is possible that in case of the Tischenko method the electrical conduction model has better perspective to be built successfully. As this method treats of the whole body, it would be possible to neglect the thorax or to replace it with a simple model. But the haemodynamics still will cause difficulties: if one does not know the minimal value of the pulsing flow (as it occurs in case of any peripheral region), it will be impossible to estimate the mean value of the flow (but just the mean value quantifies the transport function of the flow) by its pulsation only.
In the recent years a new technique has been developed on the basis of impedance plethysmography. It is named electrical impedance tomography (EIT) (Price 1979, Murai et al. 1985, Brown et al. 1986, Newell et al. 1988). The EIT technique was initially proposed for getting images of the interior of the human body. But in addition it makes possible to record time courses of local resistivities inside the body (Eyüboglu et al. 1989). The latter property of EIT would enable to overcome the described weakness of the impedance plethysmography.

EIT constitutes a sophisticated combination of multi-electrode (16 or 32 electrodes, for instance) impedance measurement and computation. There are several versions of the technique. One of the most successful versions (Eyüboglu et al. 1989) looks as follows.

The electrodes are placed around the body in a single plane onto its surface. A current source is connected between an adjacent pair of electrodes. Potentials of the other electrodes are measured then. After that the current source is connected to the next pair of electrodes and again similar measurements of potentials are done. Such shifting of current source with the consequent measurement of the potentials of all other electrodes is repeated until all the pairs of adjacent electrodes have passed through. This procedure is called a data cycle. Data sets recorded in several successive cycles are averaged to improve the signal-to-noise ratio. An averaged data set is called a frame and a single image can be built from these data. The equipment enables to record data for 24 images per second.

The recorded data are processed afterwards. The processing consists of estimation of heart cycles, elimination of breathing influence by averaging corresponding data over a certain amount (at least 100) heart cycles. Then a computational procedure called *image reconstruction* is carried out. This is a very complicated computation (see Yorkey et al. 1987) reasoning from the described above direct problem of electrical conduction in a complex region. But instead of the direct problem here an *inverse problem* (estimation of resistivity distribution from the potential distribution on the surface of region) is solved. The result is a time series of images showing time variation of conductivity distribution in the cross section of the body.

In the present stage of development EIT has spatial resolution not better than 10% of the diameter of the object. Though the resolution is low for the image processing, the result significantly improves localisation of measured impedance. Therefore EIT would enable to overcome excessive integrity of the impedance plethysmography.
Applying EIT to SV and blood flow measurement would enable better determination of the regions and organs which impedance is measured. This would clarify the situation described above in connection with the existing impedance-plethysmography-based methods and would enable to build workable conduction and haemodynamics models for the measurement. Thus it would be possible to develop better methods for SV and blood flow estimation.

Conclusion

In biomedical measurement there still stays a gap: there is no reliable and convenient non-invasive method for stroke volume monitoring in clinic. Electrical impedance methods are able to fill this gap. A modern-day method for continuous non-invasive monitoring of the stroke volume would be built on the basis of the electrical impedance measurement. However, it is essential that automation or computer assistance of the existing methods (the Kubicek's, Sramek's and Tischenko's ones) is not enough to obtain a new quality in this field. As the measurement technique as well the signal processing and interpretation would be rearranged for this purpose.

The electrical impedance tomography technique has been developed from the impedance plethysmography technique during last decade of years. This technique would be a component of the basis for new methods of stroke volume estimation. Computer simulation of cardiovascular system would be the second component of this basis and advanced signal processing would be the third.

References


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