SEGMENTAL ANTHROPOMETERIC STUDY
IN JUVENILE RHEUMATOID ARTHRITIS

A THESIS
Submitted for the Partial Fulfilment
of M. Sc. Degree

IN RHEUMATOLOGY AND REHABILITATION

BY

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Introduction
and
Aim of The Work
INTRODUCTION

Juvenile Rheumatoid Arthritis (JRA) is the most common connective tissue disease accruing during childhood. It is characterized by chronic synovial inflammation and hyperplasia (Simmons and Nutting, 1989).

The effects of diseases in childhood are similar to the effects of malnutrition (Sinclair, 1978).

As in other chronic diseases of childhood, interference with growth may occur as a part of the general metabolic disorder. However, local disturbances of growth may rise from lowering or more rarely, acceleration in the region of the affected joints (Ansell and Bywaters, 1956).

Growth retardation may also occur in selective areas, such as in jaw. Micrognathia is caused by failure of normal development of the temporomandibular growth centers or shortened finger from early hand involvement (Nelson et al., 1992).

AIM OF THE WORK

1- To identify the patterns of growth among children with juvenile rheumatoid arthritis as reflected on segmental growth.

2- To compare the diseased group with normal controls.
Review
Of
Literature
REVIEW OF THE LITERATURE

GROWTH AND DEVELOPMENT :
------------------------------

Growth is a word, a term, a notion covering a variety of diverse and complex phenomena. The terms growth, development, and maturation are often used synonymously but they are not identical. Development is a superordinate concept which subsumes growth and maturation. All are biological ideas associated with the organization of life process and living structures. Development refers to a progression of changes, either quantitative or qualitative, that lead from an immature state to a highly organized, specialized and mature state. Developmental changes are age related, they consist of transformation in the structure and function of an organism. (Prechtl, and Connolly, 1981).

The terms growth and development are often used interchangeably, and it is certainly true that each depends on the other for fruition. In the normal child each parallels the other, and any separation would be an artificial one. For convenience, however, we may distinguish between them. We restrict, when possible, the term growth to mean an increase in physical size of the whole body or any of its parts. Growth, therefore may be measured in terms of inches or centimeters and pounds or kilograms. It can be
measured also in terms of metabolic balance, i.e., retention of calcium and nitrogen by the body.

Development is used to indicate an increase in skill and complexity of function. The individual develops neuro-muscular control and character. Maturation and differentiation are frequently used as synonymous for development. It is evident that development is related to growth but is not the same (Lowery, 1986)

DYNAMIC NATURE OF GROWTH.

Growth and development are continuous dynamic processes occurring from conception to maturity and take place in an orderly sequence that is approximately the same for all individuals. At any particular age, however, wide variations among normal children reflect the responses of growing individuals to numberless hereditary and environmental factors (Rimpe and Silver, 1980).

Not all tissues of the body grow at the same rate nor stop growing simultaneously. Some tissues continue to grow throughout life time, while others reach full development early and remain relatively static thereafter (Tanner, 1978)
Periods of growth:

The growth periods were arranged in the following

A) Prenatal Period: from 0 to 280 days.

Intrauterine life is the major period of maternal influence on growth (Marton., 1955)

1- Ovum stage: From 0 to 14 days.

The stage of the ovum is characterized by increase in complexity and cell multiplication with little increase in total size.

2- Embryonic stage: From 14 days to 9 weeks.

During the embryonic period rapid differentiation takes place and all of the organ systems are established.

3- The Fetus stage: From 9 weeks to birth.

During fetal life from 9 weeks to birth, there is further development, and early functional activities are apparent. Most pronounced in the rapid increase in the body mass (Lowery., 1986).

The increase in body mass is extremely rapid with a peak rate at the month of gestation (Brook. 1978).

B) Postnatal period:

Growth during this Period is slower than during the antenatal one. It involves the stages of neonate,
II) Neural control:

It has been suggested that there may be a growth center in the brain, possibly in the hypothalamus, which is responsible for keeping the child on his genetically determined growth curve whenever possible and interacts with the anterior lobe of the pituitary gland in the hormonal control of growth. The peripheral nervous system may also play some part in the control of growth by exerting a nutritive or trophic effect on the structures they supply to modify the growth and repair the pattern of the structures innervated (Sinclair, 1978)

III) Hormonal Factors:

There are many hormones which are of particular importance in the control of growth; these are the thyroxin from the thyroid gland, the cortisol and adrenal androgens from the cortex of the adrenal gland, the testosterone from the leyding cells of the testis, the oestrogens from the ovary; the insulin from the islets of langerhans of the pancreas; the melatonin from the pineal gland. A series of hormones from the pituitary gland are also involved, the growth hormone (G.H.); thyroid stimulating hormone (T.S.H.); the adrenocorticotropic (A.C.T.H.); and the gonadotrophic hormones, which include the follicle stimulating hormone (F.S.H.); that stimulates the growth of the primary
follicles of the ovary and the sperm-producing cells of the testis, and the luteinizing hormone (L.H) or leyding-cell. stimulating hormone, which stimulates the growth and secretion of the corpus luteum in the ovary and the leyding cells in the testis. The pituitary also produces the prolactin, necessary for lactation, and the vasopressin, which is necessary for the control of body water. Another gonadotrophic hormone with actions similar but not identical to those of the L.H is produced by the cells of the chorion, which is the cell layer of the placenta nearest to the maternal circulation. The placenta also produces a hormone somewhat resembling the growth hormone, called the placental lactogen. Many hormones traveling in the blood are bound to carrier proteins. The amount of carriers may be altered by another hormone, which this influences the first hormone. The degree to which dissociation occurs may also be important, usually it is only the free portion of hormone that is active. (Prader, 1984).

A) The growth Hormone:

The growth hormone is a polypeptide secreted by the pituitary gland in a pulsatile way. It travels from the pituitary gland to all the cells of the body, where it binds to specific receptors which are widely distributed (Hughes, and Friesen, 1985).
The growth hormone maintains the normal rate of synthesis of proteins in the body, and it also inhibits the synthesis of fat and oxidation of carbohydrates. It is necessary for the proliferation of the cartilage cells in the epiphyseal plates, and so exerts a great effect on height. In contrast, it has a little apparent effect on the maturation of the skeleton, (Sinclair, 1978).

B) Somatomedin - C (I.G.FS):

Another class of growth promoting substances are called the somatomedins or insulin-like growth factors (I.G. Fs).

There is currently some debate about the relative roles that the G.H. and I.G.Fs play in cell division and growth. The I.G.Fs are similar to insulin in molecular structure and in biological action. (Preece, 1986).

Growth hormone needs an intermediary in its action on the bones to make them grow, this intermediary is called somatomedin-c (SMC) (Bionox, et al, 1986).

C) Insulin:

The insulin is a major growth regulating hormone and together with somatomedins they influence the complex growth of the fetus. It regulates the supply of substrates to the cells of metabolism (Underwood, 1984). Diabetic
children whose diabetes is well controlled by injected insulin and a suitable diet grow quite normally, but even a small degree of laxity in the control produces stunting and retardation of growth, (Tanner, 1990)

D) Thyroid Hormones:

Thyroid hormones are needed for the normal growth in stature, the development of normal body proportions, the formation of bone from cartilage, and the formation of teeth. A deficiency of these hormones (Hypothyroidism) during infancy and childhood results in growth retardation, mental impairment and in extreme cases the child suffers from a form of dwarfism called cretinism, (Bogin, 1988). Thyroxin is essential for the normal growth and development of the skeleton and of the central nervous system. It is also essential for the complete expression of the; growth hormone effect on cartilage and bone formation (Sizonenko, 1978).

E) Androgens and testosterone:

Androgen and testosterone cause increase in the muscle mass, acceleration of bone growth and maturation, growth and maturation of sexual organs. Androgens produce rapid fusion of epiphyses and cessation of linear growth (Rallison, 1986).
F) The Oestrogen:

The oestrogen increases the basal growth hormone level but it is an inhibitor to the somatic growth. It accelerates the lateral growth of the pelvis and thereby widens the hips (Vanden Brands et al. 1974). The oestrogen accelerates osseous maturation without stimulating linear growth as much as testosterone (Rallison, 1986).

G) Glucocorticoids:

Cortisol (also called hydrocortisone) and, to a lesser extent, corticosterone, is a hormone secreted in a pulsatile manner throughout life. Cortisol increases the formation of glucose from protein and has an anti-inflammatory and antistress action to 2 or 3 times the ordinary level in response to the stresses of infection, extreme exercise or severe emotions. The secretion is controlled by A.C.T.H. from the pituitary, and A.C.T.H is itself released by the action of corticotropin releasing factor from the hypothalamus. The administration of cortisol, or its near relative, to persons whose pituitaries have been destroyed is essential if they are to lead anything approaching normal lives. Cortisol is secreted during fetal life and throughout childhood in much the same amount as in adult, relative to body size. Cortisol does not have an antigrowth action. In normal amount it probably
plays no part in controlling rate of growth, but if given in excess of normal it shows up growth in height and retard skeletal maturity, while at the same time causing increase in fat. This may easily occur in children treated with corticoids for severe asthma, rheumatoid arthritis still's disease, kidney disease, or severe eczema. There is evidence that giving corticosteroides every other day instead of every day has a considerably lesser effect on growth and often suffices therapeutically. Cortisol appears to block the ability of growth hormone to produce somatomedin and this can not be overcome by giving excess growth hormone. Cortisol may also directly inhibit the action of somatomedin on cartilage cells and perhaps the secretion of growth hormone itself (Wales, and Milner, 1988).

Tomminga et al (1992) observed retardation of the height in children with acute lymphoblastic leukaemia received corticosteroid therapy. Also, he observed that, the retardation of arm span was significantly larger than the retardation of sitting height. He concluded that, corticosteroid medication is the most explanation for his finding.

H) Parathyroid Hormone:

The parathyroid hormone can influence growth and development through its effect on the mineralization of bone (Watson and Lowery; 1967).
IV) Environmental Factors:

A) Prenatal Environment

In normal intra-uterine growth, both the genotype and the environment are important. The parity of the mother, her age and the number of fetuses in the uterus influence the weight of the child at birth. The fetus may be damaged in utero by harmful agents acting upon it either directly or indirectly through the mother (Marshall, 1977).

Mechanical factors, particularly abnormalities of fetal, position and oligohydramnios, are also responsible for such congenital malformations as clubfoot, congenital bowing of the legs and microganthia (Chapple and Davidson, 1971).

B) Postnatal Environment:

1- Nutrition:

2) Socioeconomic Factors:

Physical growth was found to be significantly associated with expenditure on food (Rona, 1981). Sanitary conditions in the home (Koopman et al, 1981), mother's age, birth interval between surviving children, and family size (Goldstein, 1971) level of parental education and newspaper reading, family income and socioeconomic status in general, (Christiansen et al, 1975). Hence children in the developing countries are shorter and lighter than in advanced countries (El-Lozy, 1978).

3) Altitude:

Moderate hypoxia among children living at high altitude exerts a primary growth limiting effect even on healthy, well nourished children (Schutte et al; 1982).

4) Season:

Most European and American data show a seasonal variation of growth in height is faster in midwinter and early spring, while it is slowest at midsummer and early autumn. On the other hand growth in weight, is fastest in autumn (Marshall, 1977),
5) Exercise:

A certain minimum of physical activity is necessary to support normal growth. Physical exercises affect stature, enhance bone density and increase muscle size, strength and endurance (Falkner and Tanner, 1978).

Female swimmers seem to be slightly taller than the average population and have a greater body size (Andrew et al, 1972). Exercise reduces the storage of depot fat and consequently may alter the shape of the body. It also causes an increase in the size of muscle fibers and not in its number (Mirwald et al, 1981). However, it is necessary to specify its amount, intensity or frequency (Mirwald and Bailey, 1984).

6) Secular Trend:

It is the variation in the rate of growth with successive generation provided that all other factors are constant (Bakwin, 1964). Over the past century or more, there has been a general trend toward increases in height and weight with each succeeding generation in the more, developed nations (Tanner, 1962). The cause is said to be the consequence of a decrease in growth inheriting factors such as poor nutrition and chronic diseases combined with more genetic out breeding in recent years (Wolanski, 1984).
7) Psychological Factors:

Severe emotional disturbances result in marked growth retardation in certain children who often have siblings of normal stature. Removal of the child from this environment may result in the occurrence of a striking catch up growth (Frasier and Rallison, 1972). Psychological problems may be produced by a great variety of physical and emotional stresses such as birth defect, physical injury, inconsistent and contradictory child rearing practices, marital conflict, child abuse and neglect and chronic illness (Nelson et al. 1992)

8) Diseases:

Almost any illness may have some defect on growth but acute minor illnesses, such as the childhood exanthematous even more severe ones, such as pneumonia, do not usually cause a discernible retardation in the growth of well nourished children. There may be a greater effect in children whose diet is less adequate but this is still open to doubt (Marshall, 1977). The effects of diseases in childhood are similar to the effect of malnutrition and good examples are afforded by tuberculosis and kidney disease (Sinclair. 1978).
Chronic diseases cause slow growth, the exact cause is not known. It may be attributed to the stress condition of the disease which leads to an increase in the secretion of cortisone and to a decrease in the secretion of growth hormone, or it may be due to an error in the body metabolism or an absorption defect (Abou EL Soud, 1984). One of the most common chronic diseases associated with stunted growth are chronic renal diseases (Hodson et al., 1983) congenital heart disease (Mehrizi and Darsh 1962 and (Khattab et al., 1985) chronic asthmatic bronchitis (Hauspie et al. 1979) liver cirrhosis (Osman et al. 1974) parasitic infestation (Jelliffe and Standfield, 1978) and juvenile rheumatoid arthritis (Ansell and Bywaters, 1956; Bernstein et al., 1977; Ansell, 1978 Cassidy, 1982; EL Awar, 1987, and Nelson et al., 1992).
JUVENILE RHEUMATOID ARTHRITIS

Juvenile rheumatoid arthritis (JRA) is a disease or a group of diseases characterized by chronic synovitis and associated with a number of extra-articular inflammatory manifestations. A number of confusing names have been applied, including juvenile arthritis, Still's disease, juvenile chronic polyarthritis and chronic childhood arthritis (Nelson et al., 1992).

Most children with JRA enter into remission and reach adulthood without significant disability, however, up to 25 percent of them have an unremitting disease which can result in significant musculoskeletal deformities and functional limitations (Schaller, 1984).

Approximately 10-15 of these children lose the ability to ambulate and become dependent on others for self care (Brewer, 1975).

The clinical presentation is extremely varied in scope and severity. Juvenile rheumatoid arthritis encompasses several broad clinical subgroups (Nelson et al., 1992).
(Table I) Subgroups of Juvenile Rheumatoid Arthritis

<table>
<thead>
<tr>
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<th>Polyarticular Rh. Fact.</th>
<th>Pauciarticular</th>
<th>Systemic onset</th>
</tr>
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<tbody>
<tr>
<td>percentage</td>
<td>negative 20-25</td>
<td>positive 5-10</td>
<td>Type I 35-40</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Type II 10-15</td>
</tr>
<tr>
<td>Sex</td>
<td>90% girls</td>
<td>80% girls</td>
<td>90% boys 60% boys</td>
</tr>
<tr>
<td>Age at onset</td>
<td>Throughout childhood</td>
<td>late childhood</td>
<td>late childhood</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Throughout childhood</td>
</tr>
<tr>
<td>Joints</td>
<td>Any multiple</td>
<td>Any multiple</td>
<td>few large joints</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>knee, ankle, hip</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>elbow, girdle</td>
</tr>
<tr>
<td>Sacroiliitis</td>
<td>No</td>
<td>rare</td>
<td>No common</td>
</tr>
<tr>
<td>Iridocyclitis</td>
<td>rare</td>
<td>No</td>
<td>30% ch. chr. irid.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>10 - 20% acute irid.</td>
</tr>
<tr>
<td>Rheumatoid factor</td>
<td>Negative 100%</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>Antinuclear antibodies</td>
<td>25%  75%</td>
<td>90%</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>HLA studies</td>
<td>?</td>
<td>HLA DR4</td>
<td>HLA DR5 DRW6/DRW8</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>HLA B27</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>?</td>
</tr>
<tr>
<td>Ultimate morbidity</td>
<td>severe severe</td>
<td>ocular subsequent severe</td>
<td></td>
</tr>
<tr>
<td>arthritis</td>
<td>arthritis arthritis damage 10% spondyloarthropathy, arthritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-15% &gt; 50%</td>
<td></td>
<td></td>
<td>25%</td>
</tr>
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30% girls m. rh. we ( 60% boys ) ?
Etiology:

The etiology and pathogenesis of JRA are unknown. JRA may not present a single disease but a syndrome of diverse etiologies. Potential factors include infections, autoimmune etiologies, somatic response to stress and heredity (Simmons and Nutting, 1989).

I) Infection:

Several studies have demonstrated antibodies to bacterial peptidoglycan particularly in patients with pauciarticular JRA (Burgos et al. 1986).

A viral etiology for JRA remains speculative with little firm data available to support this theory. Certainly the clinical picture of systemic onset JRA is suggestive of a viral infection, although this hypothesis has not been proven despite years of study. The fact that a number of viral infections are known to be associated with self limited arthritis in children adds further support to the concept of a role for viruses in JRA (Lang and Shore, 1990).

It is well recognized that intercurrent viral infection may induce flares of JRA. The mechanism of this phenomenon is unknown (Lang and Shore, 1990).
II) Autoimmune theory:

Autoimmune reactions to unknown stimuli, play a central role in the pathogenesis of RA through a complex mechanism. The immune complexes of the rheumatoid factor and immunoglobulins may precipitate synovial inflammation and are responsible for the vasculitis seen in sero-positive rheumatoid arthritis. However, this mechanism fails to explain all rheumatoid inflammations, since chronic synovitis can occur in the absence of the rheumatoid factor with a normal level of complement in the joint fluid. The occurrence of chronic arthritis in patients with IgA deficiency and hypogamaglobulinemia suggests that immunodeficiency may somehow predispose to rheumatoid arthritis (Nelson et al., 1992).

III) Familial and genetic factors:

Somatic responses to stress and heredity are cited as precipitating factors that may trigger the disease (Barry et al., 1989). Family studies of patients with juvenile chronic polyarthritis have shown an increased incidence of ankylosing spondylitis in male relatives, and of sero-negative chronic polyarthritis in female relatives. Also of sacroiliitis in male relatives aged 15 years and over, and in female relatives aged 45 years and over (Ansell et al., 1968).
The relatives of patients with still's disease not only have a greater incidence of clinical inflammatory polyarthritis, but radiologically, they also show a greater frequency of sacroilitis and of erosive arthritis in the hands and feet (Barbora et al. 1962).

Pathology:

JRA is considered to be the juvenile form of rheumatoid arthritis although it differs in several respects from the adult form (Sokoloff, 1979).

Articular lesions:

---

Synovitis:

---

The microscopic appearance of the synovial tissues are oedematous, hyperemic and infiltrated with lymphocytes and plasma cells. Thickened synovial membranes form villi which protrude into joint space (Nelson et al., 1992).

Articular cartilage:

---

The articular cartilage is eroded and progressively destroyed, the mechanism of destruction of articular cartilage and other joint structure by chronic proliferating synovium remains unknown. Joint destruction occurs more often in children with rheumatoid factor positive or systemic onset disease (Nelson et al., 1992).
Subcondral bone:
---------------

Once joint destruction has commenced, erosions of subcondral, narrowing of the joint space, fusion of bones, and deformity, sublaxation, or ankylosis of joints may occur.

Subcutaneous nodules:
---------------------

The rheumatoid nodules are less frequent in children with JRA (Nelson et al., 1992).

Ocular changes:
---------------

The uveitis "iridocyclitis" does not have distinctive clinical characteristics. Band-shaped keratopathy, a characteristic equatorial corneal opacity due to deposition of calcium beneath the corneal epithelium is a sequelae of the iridocyclitis (Henkind and Gold, 1973).

Cardiac lesions:
-----------------

Pericardial involvement occurs in the form of non-specific fibrous serositis. It may be detected clinically with greater frequency than in adults (Lilman and Bywaters 1963).

Skin eruptions:
---------------

The erythema is of transient form. Histologically,
only mild perivascular aggregation of mononuclear cells in the superficial dermis has been observed (Isdale and Bywaters, 1956).

Clinical picture:

The clinical onset of the disease may follow an acute systemic infection or a physical trauma to a joint, but no direct relation has been shown. Exacerbation may follow intercurrent illness or physical stress (Nelson et al 1992).

Girls account for the majority of cases in the early peak of the distribution curve and are affected twice as boys except for systemic onset in which the sex ratio is equal. The most obvious manifestations in children with JRA may be, increased irritability, assumption of a posture of guarding the joints or refusal to walk. Fatigue and low grade fever are common at onset. Anorexia, weight loss and failure to grow are seen in many children (Cassidy, 1989).

Three major subgroups of JRA are recognized by clinical findings, laboratory data and presentation at onset.

JRA is classified as follows.

1- Systemic onset:

Still's disease is a clinical entity of unknown
origin which can appear before 15 years of age (Juvenile onset still’s disease) or later (adult onset still’s disease). It is concluded that the articular prognosis of still’s disease is poor, be it adult onset or juvenile onset, with severe joint destruction in half of the patients. The only difference between juvenile onset still’s disease and adult onset still’s disease is the occurrence of amyloidosis which is restricted to adult onset still’s disease. (Cabane et al 1990)

About 20% of all JRA patients have systemic onset disease. Boys and girls are equally affected or there may be slight male preponderance (Schaller and Wedgwood, 1972)

The most distinguishing features of systemic onset JRA are a daily spiking fever, with a return to baseline temperature at least once a day, and a rash. The rash consists of salmon-pink maculopapular lesions, which are about 1 cm in diameter. The papules occasionally coalesce into large confluent lesions which are marked at the height of the fever. The rash can be missed if it is not looked for at the peak of the fever (Baum, 1990).

Rheumatoid rash may occur anywhere on the body especially on the trunk and proximal extremities (Calabro and Marchesano, 1968).
Enlargement of the spleen is most prominent within the first year of disease. Lymphadenopathy in the cervical, axillary and epitrochlear areas in usually symmetrical (Cassidy, 1989)

Most children with systemic onset JRA have arthritis within a few months of onset, while a few patients do not develop arthritis until years later. The pattern of joint involvement is ultimately polyarticular. Systemic manifestation rarely recur when patients reach adulthood, even although chronic arthritis may persist (Nelson et al., 1992).

Polyarticular onset :

Two subgroups are included: Rheumatoid factor negative polyarthritis (25% of total JRA patients) and rheumatoid factor positive polyarthritis (10% of total JRA patients) (Nelsoner et al., 1992). Hidden IgM and IgG rheumatoid factors can be detected by different techniques in children who are seronegative by classic agglutination methods (Moore et al., 1986).

Polyarticular seropositive JRA have its onset in late childhood with more severe arthritis, frequent rheumatoid nodules and occasional rheumatoid vasculitis. Polyarticular seronegative JRA may begin at any time during childhood, arthritis is mild, and is rarely associated with rheumatoid
nodules. More girls than boys are affected in both types. Affected joints are swollen and warm, but rarely red, swelling results from periarticular oedema, joint effusion, and synovial thickening. Early in the disease limited joint motion is related to muscle spasm, joint effusion, and synovial proliferation, later limited motion may result from joint destruction and ankylosis or from contractures of soft tissue. Pronounced synovial proliferation may produce cystic swellings about affected joints. Young children with multiple joint involvement, are often irritable and assume a typical posture of anxious guarding of their joints against movement (Nelson et al., 1992)

The onset is insidious with progressive joint involvement. The arthritis generally involves the knees, wrists, elbows and ankles, but may include small joints of the hands and feet. Involvement is symmetric (Cassidy, 1989).

Neck involvement is common, usually affecting the upper cervical spine. The neck is painful and stiff with rapid loss of extension and rotation. Later fusion of the apophyseal joints as well as the bodies occurs (Ansell and Bywaters, 1978) Temporomandibular joint involvement with limited ability to open the mouth is common, hip involvement usually begins later in the disease process (Nelson et al., 1992)
Patterns of joint involvement in seropositive disease do not differ from those of seronegative polyarthritis except for less frequent involvement of distal interphalangeal hand joints, rapid progressive course and destructive arthritis. (Schaller, 1986). Systemic manifestations in polyarthritis are less acute than in children with systemic onset, low grade fever, malaise, anorexia, irritability and mild anemia, slight hepatomegaly and lymphadenopathy may be present (Cassidy, 1989).

3- Pauciarticular onset:

Pauci articular onset JRA is the most common type, about 50% of juvenile arthritis. (Baum, 1990)

The onset involves four or fewer joints, large joints are primarily affected, the distribution is often asymmetrical two subgroups are included:

A) Pauciarticular Type I:

Consists of patients with disease onset in early childhood (before age of 4 predominantly girls who often have positive tests for ANA and frequently develop chronic iridocyclitis (By waters and Ansell, 1965).

The most commonly affected joints are the knees, ankles, and elbows, there is spotty involvement of other joints such
as the T.M. joint, single toes or fingers, wrists or neck. The test for rheumatoid factor is negative, If arthritis remains limited to a few joints for the initial 6 months, the disease remains pauciarticular through its course, one or both eyes may be affected, if initial involvement is unilateral, the other eye usually remains uninvolved (Nelson et al., 1992) Systemic manifestations have not been prominent (Cassidy et al., 1967).

2) **Pauciarticular type II:**

includes a group of children who are not at risk of chronic iridocyclitis and do not have positive test results for ANA (Schaller, 1980). Males are more affected, the age of onset is generally after 8 years old (Ansell, 1978). The test for the rheumatoid factor is negative, 75 percent of patients have HLA B27, large joints are affected. The foot joints, T.M Joints and joints of the upper extremities are also involved (Nelson et al., 1992)

**Extra-articular manifestations of JRA :**

**Cardiac involvement :**

Cardiac involvement in JRA is well documented. It occurs in JRA as it does in adult rheumatoid arthritis. Heart disease in JRA is usually confined to the pericardium and myocardium while valvular involvement is rare (Svantesson et al, 1983 a). In radiographic examinations, cardiomegaly
may be present particularly if myocarditis or pericardial effusion is present. In some instances, cardiomegaly may be related to anemia rather than cardiac disease (Moller and Pierpont, 1983).

Pericarditis is a common non-articular manifestation in JRA. It occurs most frequently in the systemic subset of the disease (Ansell and Bywaters, 1986).

Sevantesson et al, (1983), found that abdominal pain and vomiting were common in patients with Pericarditis indicating a concomitant aseptic peritonitis. Also pleuritis was often associated with pericarditis as a part of general polyserositis. Each episode of Pericarditis generally persists for a week to 4 months (Cassidy, 1989).

Pulmonary involvement:
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Pulmonary disease is rare in children with JRA, though pulmonary fibrosis has been reported in seropositive cases (Jordan and Snyder, 1964).

Renal involvement:
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Renal glomerulitis may be a part of the clinical course of the disease and can present real problems of differential diagnosis relative to analgesic administration or gold therapy. Forty five percent of children with JRA
may show abnormalities on urine analysis (Antilla, 1972)

Subcutaneous nodules:
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Subcutaneous nodules are associated with rheumatoid factor seropositivity, and are typically seen in patients with polyarthritis (Kaye et al, 1984).

Eye involvement:
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Chronic uveitis is especially likely to occur in girls of early age of onset of disease with limited joint disease, who have ANA positivity (Schaller et al, 1974).

The onset is usually insidious and asymptomatic so routine ophthalmologic examination must be performed at the time of diagnosis in every child with JRA and should be repeated at frequent intervals during the first year of the disease. The only hope for saving the eye sight in the affected children is early and persistent use of mydriatic and corticosteroid drugs (Cassidy, 1989).

Investigation
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I- Laboratory investigations:

Laboratory tests can be helpful in establishing the diagnosis or in subgroup classifications, however, no single test or group of tests are diagnostic. (Butler, 1968).
Many children develop a normocytic, hypochromic anemia during periods of activity of the disease. Leucocytosis is common with active disease especially in children with systemic onset (Cassidy 1989). The platelet count is positively correlated with disease activity (Balogh et al., 1980).

Acute phase reactants are elevated in active JRA and provide an index for disease activity and response to therapy, the erythrocyte sedimentation rate is most commonly used, (Joseph and Buther, 1986).

Rheumatoid factors are found in about 5% of children with JRA and correlate with older age at onset. Positive test results are most commonly associated with polyarticular disease late childhood onset, severe destructive arthritis, and rheumatoid nodules, rheumatoid vasculitis, and Sjogren syndrome are also occasionally associated (Nelson et al., 1992).

Anti-nuclear antibodies are positive in 40% of children with JRA (Petty et al., 1973). The frequency of ANA increases in girls of younger age of onset and decreases in older boys and in children with systemic disease. They reach their highest prevalence in children who have oligoarthritis and uveitis (Schaller et al 1974).
Lupus -Erythromatosus (LE) cells are seen in a very small number of children with classic JRA (Schaller and Wedgwood, 1972).

The presence of HLA-B27 is characteristic of the spondyloarthropathies (Hall et al., 1975).

The synovial fluid examination in JRA is cloudy, may clot spontaneously, and usually contains increased amounts of protein. The level of glucose may be low in the joint fluid, while the level of complement may be normal or decreased (Nelson et al., 1992). Also, the white blood cell count does not always correlate with the degree of clinical activity, low counts have been observed in fluids from joints, clinically involved by active inflammatory disease (Zuckner et al., 1977).

Urine analysis is usually normal in children with JRA except for proteinuria associated with fever. Persistent proteinuria is often the first indication of amyloidosis. Intermittent haematuria may be an evidence of glomerulitis associated with JRA, drug toxicity, or the development of another disease such as lupus (Cassidy 1989) There was significant relation between haematuria and disease activity (Osdogan, et al. 1987).
II- Radiological examination:

Radiological examination in JRA consists of soft tissue swelling, osteoporosis, and periostitis about affected joints. Regional epiphyseal closure may be accelerated and local bone growth increased or decreased in long active joint disease subchondral erosions and narrowing of cartilage spaces may occur (Nelson et al., 1992).

In the neck, Atlanto axial subluxation is the most characteristic change in the cervical spine. Apophyseal joint disease of the upper cervical segments occur and bony fusion is frequent. Vertebral compression fractures are common as a consequence of osteoporosis of the disease process and corticosteroid therapy (Ansell and Kent, 1977).
TREATMENT

The management of children with JRA needs a team of the pediatrician, rheumatologist, orthopedist, ophthalmologist, psychiatrist, physical therapist, social worker, and others. The goals of treatment are to achieve a state where in the child can live at home like other children, attend an ordinary or special school, and remain free of contractures and gross deformities. The principles of management include basic supportive measures and drug therapy (Calabro 1979).

Basic supportive measures:
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The first step is to educate family members on the nature of the disease and their role in treatment. The child needs a well-balanced diet, while supplemental vitamins may be added for those who are not eating properly. Infections should be cared for, since these often precipitate exacerbations of the rheumatoid disease (Brewer, 1975).

I- Physical therapy:
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During periods of acute inflammation of the joints rest and range of motion in a whirlpool bath is advised.

A balanced program of increased rest at night or
during the day after school is advisable. Normal play should be encouraged.

Cock-up splints for the wrists and posterior night splints for the knees are used to prevent malpositioning of the joints.

A prone lining board will help to prevent hip flexion contractures.

For severe contractures, serial casting with active exercises may be tried.

Malpositioning of the cervical spine may be minimized by the use of a soft collar and a desk with tilted top on which the child can do his homework (Cassidy, 1989).

II- The drugs used include:

1- Aspirin:

Salicylate is the drug of choice in the initial treatment of JRA. In therapeutic dosage, it usually alleviates both arthritis and systemic manifestations (Lindsley, 1981).

The mechanisms of action of aspirin include effects on the immune response, stabilization of cell membranes and inhibition of prostaglandin synthesis (Smith, et al., 1975).
Both levels "20-30mg/dl can be reached by using doses of about 100 mg of aspirin /kg daily for children of 25 Kg or less. Child must be watched carefully for toxicity. A full therapeutic response may require weeks to months. The symptoms of toxicity include rapid, heavy breathing which are the earliest signs of salicylism in children. Also, elevated levels of hepatic enzymes have been described in the sera of children with rheumatic diseases who are receiving large doses of salicylates (Nelson et al., 1992).

Few patients may develop occult gastrointestinal bleeding occasionally dyspepsia or rash (Rich and Johnson 1973).

When children do not respond to this program after 6 months trial, or respond inadequately, other drugs can be considered (Cassidy, 1989)

2- Non-Steroidal anti inflammatory agents (NSAID<sub>i</sub>) :

The present trend in therapy is to use NSAID<sub>i</sub> other than aspirin. Non-aspirin NSAID<sub>i</sub> have the advantage of fewer tablets per day, no reports of the development of Reye's syndrome, and some reports of greater effectiveness than aspirin. (Baum, 1990).
All of NSAIDs have analgesic, anti-inflammatory and antipyretic effects and all inhibit prostaglandin synthesis. They would be indicated for control of pain, stiffness, and inflammation in selected children who have been unresponsive to aspirin. The clinical response to NSAIDs varies among individuals and lack of response to one drug does not necessarily mean that the patient will not respond to another (Lindsay, 1981).

Ibuprofen is safer than aspirin and has a generally good record (Daniel, 1992).

In children older than 14 years any NSAIDs may be tried but for children less than 14 years old tolmetin, naproxen and fenobrofen have been approved (Simmons and Nutting 1989).

3- Disease modifying antirheumatic drugs (DMARD)

* Gold salts

Two forms of gold salts are commonly used, gold sodium thiomalate and gold sodium thioglucoce (Lindsay, 1981).

Intramuscular gold preparations in a dose of 0.75 mg/kg/week have been used successfully to reduce disease
activity though the requirement for injection puts the child through some discomfort. (Baum 1990).

Oral gold agents have been studied. (Auranofin) Triethyl phosphine gold appears to be effective in a dose of 0.1 mg/kg/day, with allowable increase to 0.2 mg/kg/day (Brewer et al., 1986).

Before administration, the child’s basic hematologic kidney and liver functions must be normal. Indication for discontinuing gold include a decrease in the leucocytic count to less than 400/cubic millimeter, a 50 percent fall in the absolute neutrophil count, development of eosinophilia, proteinuria, or hematuria and mucocutaneous signs such as exfoliative dermatitis and stomatitis. (Rosenberg, 1989)

Antimalarials drugs:

The antimalarials are of uncertain value in children. Chloroquine is especially dangerous because accidental ingestion of as little as 1 gm. can produce rapid death. No known antidote exists; survival depends on prompt endotracheal intubation and gastric lavage (Calabro, 1979).

Hydroxy chloroquine is probably the safest DMARD (Daniel, 1992). It may be a preferred form because
retinopathy appears to occur less frequently than with other antimalarial drugs (Maksymowych and Russel, 1987). A dose of 5 to 7 mg/kg/day is recommended (not to exceed 200 mg/day), after 6 to 8 weeks dosage should be reduced and the total period of initial treatment should not exceed 2 years (Laaksonen et al., 1974). Eye examination be performed before the initiation of antimalarial therapy and at 3 months interval thereafter. Absolute indication to discontinue the drug includes documented aberration of vision, especially abnormalities of colour discrimination indicative of foveal involvement (Rosenberg, 1989).

**Penicellamine:**

The dose is 10 mg/kg/day (Brewer et al, 1986). Complete blood count and urine analysis are done once a week. Important side effects are drug induced lupus, dermatitis, thrombocytopenia and proteinuria (Cassidy, 1989). Brewer et al., (1986), failed to demonstrate significant improvement in comparison with placebo.

**Sulfasalazine:**

The encouraging results described with sulfasalazine in adult rheumatoid arthritis and in adult spondyloarthropathies, stimulated us to use the drug in children with JRA (Stefan et al., 1991).
Sulfasalazine has been used in children with JRA as well as in those with the arthritis of inflammatory bowel disease (Cassidy, 1989).

A clinical response was observed within 3 months of treatment, but in some patients clinical remission is obtained only after 6 to 12 months, so Sulfasalazine seems to have a slow acting effect on disease manifestation in JRA. Child in whom a prolonged remission was achieved, No recurrence of the disease was observed after discontinuing the drug. Sulfasalazine seemed to be more effective in pauciarticular than in polyarticular forms (Stefan et al., 1991).

The toxicity of sulfasalazine treatment was gastrointestinal intolerance, leucopenia, rash, and nervous agitation. These side effects were all reversible after discontinuation of the drug (Stefan, et al., 1991).

Immunosuppressive agents:

The drugs most commonly employed are azathioprine, cyclophosphamide, chlorambucil, and methotrexate. Leucopenia or bone marrow suppression may result from any of these agents. The potential for development of malignancy and mutagenic effects are the most critical long term considerations. (Cassidy 1989).
Newer immunoregulatory drugs such as levamisole must be approached with great caution in the treatment of children with JRA. Several children treated with this drug developed fatal agranulocytosis or seizures (Prieur et al., 1978).

Methotrexate (MTX) is well tolerated and efficacious in treatment of refractory JRA (Rose et al., 1990).

The full spectrum of its toxicity is not yet completely defined, reports of acute (Although transient ) hepatic failure have recently surfaced, as have reports of serious fungal infections. Likewise, previously unrecognized CNS toxicity and significant weight loss have been documented. The major point to be made here is that the use of MTX in JRA must not be embraced without an appropriate awareness of its potential toxicity (Furst, 1990).

In considering the use of azathioprine in JRA, the authors indicated that this drug may be toxic at 2.5 mg/kg/day. (Daniel, 1992).

An open trial of cyclosporine in JRA resulted in significant toxicity, requiring discontinuation, this drug cannot at present be recommended (Ostensen, et al., 1988).

Chlorambucil in a dose of 0.15 mg/kg /day may be indicated in severe unresponsive iridocyclitis (Mehra et al., 1981).
In a study by Bjerkhoel and Forre 1988, a patient with early onset systemic JRA who did not respond to therapy with systemic corticosteroid and cytotoxic drugs, recovered after cyclosporine treatment and had no remaining signs of illness.

Corticosteroids:
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Corticosteroids are the most potent anti-inflammatory agents used in the treatment of rheumatic disease. They are known to reduce vascular permeability and suppress leukocyte migration and immune response (Lindsley, 1981). Despite potent anti-inflammatory and immunosuppressive effects, steroid therapy does not favorably influence the duration or eventual prognosis of arthritis. Systemic corticosteroids may be given for life threatening, extra-articular diseases such as severe pericarditis in a child with systemic onset JRA or in a child with persistent iridocyclitis despite topical steroid therapy. For the child with severe systemic disease, oral prednisone in a dose of 0.5 to 1 mg/kg/day is a recommended initial dose but doses as high as 2 mg/kg/day may be eventually required with a satisfactory response. The dose should be gradually tapered then finally discontinued (Rosenberg 1989).

Topical steroid is the preferred form of therapy for treatment chronic iridocyclitis associated with JRA.
Intra articular steroids may be of value in the child whose disease is active in only one or two joints. The use of intra articular injections should be limited, owing to the potential for damaging articular cartilage in a growing child.

Corticosteroids should rarely be used for relief of joint manifestations alone because they neither cure arthritis nor prevent joint damage, and their chronic side effects may be even less tolerable than the joint disease (Nelson et al., 1992).

High dose intravenous pulse steroid therapy should be reserved for acute systemic disease unresponsive to more conservative therapy. Methyl prednisolone 30 mg/kg/day given in 100 ml of 5% dextrose in water infused over 3 hours is the recommended pulse steroid regimen (Miller, 1980).

There is no evidence of adrenal cortical suppression at the low dose of prednisolone, mean 5.6 mg daily (Mahoney et al., 1989).

The severity of glucocorticoid side effects depends on the time, the day of administration (Kowanko, et al., 1982). Adrenopituitary suppression is maximal when steroids are given at night but minimal or absent with morning
administration (Nichols et al., 1965). Other side effects, for example nocturia may be reduced by morning only administration (Kowanko et al., 1978).

Suppression of the adrenopituitary axis by exogenous glucocorticoids depends on the duration of therapy, the daily dosage, the time of administration and the formulation of the steroid used (Voigt and Fehm, 1980).

In diseases for which higher doses are required administration on alternative days, may be necessary to avoid Hypothalamo-pituitary-Adrenal suppression. Very potent steroids such as dexamethasone, betamethasone, or triamcino-lone may cause suppression even when given only twice weekly. They should be avoided unless essential for very high dose medication. Many studies have shown that the degree of suppression may be diminished if the corticosteroid is given in a single dose on alternate days. Treatment with glucocorticoids causes wasting of the proximal skeletal muscles. There is evidence that physical training improves muscle mass and strength. Other side effects include osteoporosis, increased susceptibility to infections, Cushing syndrome and growth retardation. Also, destruction of cartilage and aseptic necrosis of bone, particularly in the femoral heads, may be related to long-term steroid therapy (Nelson et al., 1992).
Earl Silverman, (1989) have demonstrated that intravenous gammaglobulin (IVGG) (1gm/kg/day for 2 days monthly, for at least 6 months) improves articular signs somewhat, but is more dramatic in alleviating extra articular features and abnormal laboratory values in systemic juvenile chronic arthritis. The children under the study were 3 to 10 years of age, had their disease for a mean of 1.7 years and were all taking nonsteroidal anti-inflammatory drugs.

III- Orthopedic surgery:

The purpose of synovectomy is to decrease the inflammatory activity in the joints from which the diseased synovial tissue is removed. It is desirable to perform a synovectomy before joint destruction and fixed deformity occur. Surgical interventions considered after 6 months of persistent synovitis despite good medical management. Persistent pain, large boggy and swollen joints, radiological evidence of joint destruction, and decreased range of motion are indications for synovectomy. (Barry, et al., 1989)

Tenosynovectomy may be indicated to decrease the risk of tendon rupture over the dorsum of the wrist or for adhesive tenosynovitis and trigger finger (Cassidy, 1989).
Course and prognosis:
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The patient with JRA should be treated as normally as possible by family, teachers, and friends. Regular physical activities, should be encouraged and incorporated into the total therapeutic program. The majority of children do not suffer long term disability and are able to function normally as adults.

The major cause of morbidity in polyarticular and systemic JRA is chronic joint disease, in pauciarticular disease, the major morbidity is chronic iridocyclitis in type I patients and subsequent spondyloarthropathy in type II patient. Even with severe systemic involvement, the disease is rarely life threatening. Some patients continue to have active arthritis into adulthood, and some have exacerbations after many years of apparently complete remission, exacerbations may be associated with intercurrent illness, hepatitis, and other forms of liver disease may be followed by transit remission of arthritis.

Patients with rheumatoid factor-positive polyarthritis and systemic onset disease have the poorest prognosis for joint function. Seventy five percent of patients with JRA have long remissions without significant residual deformity or loss of function (Nelson et al., 1992)
Hip disease is a major cause of disability in JRA and is interpreted as a relatively poor prognostic sign (Bernstein et al., 1977).

Osteoarthritis may occur later in the course of the disease after clinical remission of active inflammation, particularly in the weight bearing joints.

About half the children with systemic onset with recover completely, the other half continue to show progressive polyarthritis and moderate to severe disability. The prognosis for sight in children with chronic uveitis is probably improving because of earlier detection and better management.

Amyloidosis usually develops in more severely affected children with disease activity of long duration generally heralded by proteinuria and diagnosed by rectal biopsy.

Death occurs in 2 to 4 percent of children with JRA the major causes are renal failure, in half the cases associated with amyloidosis, and infections (Bernstein, 1977).
GROWTH IN JUVENILE RHEUMATOID ARTHRITIS

Growth in health is a very regular process. Children have their trajectories, governed by the control systems of their genetical constitution and powered by energy absorbed from the natural environment. Deflecting the child from its growth trajectory by acute malnutrition or illness, and then a restoring force develops so that, the missing food is supplied or the illness, terminated, the child catches up towards his original course and when he gets there, the velocity of growth shows down again to adjust his path on the old trajectory once more (Prader, et al., 1963).

In common with other chronic disease states, children with JRA show growth retardation. This is independent of the local effects, which may result in premature epiphyseal fusion and bizarre growth abnormalities, but is dependent on the duration and activity of the illness, only those with prolonged disease are developing severe residual height defects (Ansell and Bywaters, 1956).

Disturbances of growth are characteristic features of arthritis in children (Larheim et al., 1981)

Because chronic inflammatory arthritis causes prominent systemic effects and occurs at areas of very active growth,
JRA may cause generalized impairment of growth (Edmondo and Hughes, 1985).

Impairment of general growth and development occurs in half of all JRA patients (Cassidy, 1989). This is related to active disease as well as to prolonged daily corticosteroid administration, even in dosage as low as 3-5 mg/day prednisone (Ansell and Bywaters, 1986).

During periods of active disease linear growth is usually retarded (Bernstein et al., 1977).

When the disease becomes inactive, growth is resumed and the child reaches normal proportions rapidly. On the other hand, if disease activity is prolonged, permanent stunting of growth may occur from early closure of epiphyses (Calabro, 1979).

Growth disturbances adjacent to inflamed joints may result in either overgrowth or undergrowth of the affected part, however growth spurts often occurs with remission (Nelson et al., 1992).

Growth retardation may also occur in selective areas, such as in jaw. Microganthia is caused by failure of normal development of the temporomandibular growth centers or
results from involvement of the cervical spine (Sairanen, 1970 and Larheim et al 1981). The most extreme degrees of micrognathia are due to disease of long duration that had its onset before the fourth birth day (Cassidy, 1989).

Secondary growth deformities are frequent in children with limited joint disease involving the knee and result in unequal leg length. The involved leg usually grows longer (Cassidy, 1989).

Early during active disease, development of the ossification centers or epiphyses may be accelerated and later stunting, dwarfing, and premature fusion of the involved bone may result (Martel et al 1962).

The most common local growth abnormalities are due to premature appearance of epiphyses in the young and premature fusion in the older child. This is particularly common in the metacarpals, metatarsals and in the mandible (Calabro, 1979).

So, increased leg length may follow chronic arthritis of the knee, and micrognathia after tempromandibular arthritis may be a late hallmark of JRA. Small deformed feet may result from foot involvement in early childhood and

Brachydactyly develops from premature closure of the epiphyseal growth plates (Cassidy, 1989).

As a result of fusion of the epiphyses in one involved joint, one limb may be shorter than the other (Ansell and Bywaters, 1978).

Local growth abnormalities in the areas of affected joints are probably due to alteration in blood supply, and perhaps to prolonged steroid administration (Bache, 1964).

Not only does Juvenile chronic arthritis appear to impair linear growth in children, but that growth can be further compromised by corticosteroids (Davies, 1989).

So impairment of growth may be further aggravated by corticosteroids administration. Byron et al., (1983) observed that, linear growth of children with juvenile chronic arthritis were clearly suppressed and failing to grow in children receiving daily corticosteroid therapy, but not suppressed and growth was satisfactory in children receiving alternate day therapy. He suggested that daily divided doses of corticosteroid should not be used. Ideally a single morning dose (up to 2 mg/kg) alternate-day regimen should
be employed, as this regimen has minimal effects on hypothalamic-pituitary adrenal axis function and growth, even in young children.

So although the dose and duration of corticosteroid therapy are important (Blodgett et al., 1956), the regimen of administration and timing of the dose appears to be equally important. Children on alternate day regimens, but taking their steroid in the evening or in divided doses in the alternate day, have been suppressed and failing to grow (Byron et al., 1983).

In a child receiving alternate day oral steroids, the growth occurs in day free of treatment (Wales and Milner, 1988).

Very potent steroids such as dexamethasone, betamethasone or triamcinolone may cause suppression even when given only twice weekly (Rabhan, 1968).

The mechanism of the growth inhibition due to steroid administration is uncertain but is probably secondary to a peripheral blocking of the action of the growth hormone (Preece 1976). Or may be due to direct action on the target tissues and by the inhibition of the growth hormone action (Underwood and Kenan, 1981). Also may be due to
suppression of the secretion of growth hormone (Bozzola et al., 1991).

The suppression of growth may be reversed and catch up occur by discontinuing the corticosteroid or after completion of treatment (Blodget et al., 1956 and Tamming et al., 1992), or changing from a daily to an alternate-day regimen with either prednisone (Soyka, 1967 and Guest and Broyer 1991).

Catch up of growth occur only in prepubertal or undergoing puberty children (Guest and Broyer, 1991). Recovery does not always occur, and some patients appear to have a persistent retardation of growth (Norman and Sonders, 1969).
Material and Methods
PATIENTS AND METHODS

This study comprised 70 children attending the outpatient clinic of Banha and Kasr El-Eini University hospitals. These children were classified into two groups:

**Group I**

Included 40 patients (27 females and 13 males) ranged in age between years, three years and one month to eighteen a with mean age of 11.85 years. They were suffering from JRA diagnosed according to the American Rheumatism Association criteria (Brewer et al., 1986).

1- Age of onset less than 16 years.

2- Arthritis in one or more joints defined as swelling or effusion, or by the presence of two or more of the following signs: limitation of range of motion, tenderness or pain on motion, and increased heat.

3- Duration of the disease from 6 weeks to 3 months.

4- Type of onset during the first 4 to 6 months is classified as:

a] Polyarthritis: when 5 joints or more are involved.

b] Oligoarthritis: 4 joints or more in involvement.

c] Systemic disease: Intermittent fever, rheumatoid rash, arthritis, visceral disease (hepatosplenomegaly, lymphadenopathy, etc.).

5- Exclusion of other causes of arthritis.
Group II:

Included 30 healthy, children (19 females and 11 males) ranged in age between three years and one month to eighteen years with a mean age of 10.97 years. These subjects were considered a control group.

METHODS

All the patients were subjected to full history taking, thorough clinical examination and routine investigations to confirm the diagnosis, according to the following:

I- History taking:

A] Personal history:

Name, Age, sex and residence.

B] Complaint:

In the Patient’s own words

C] Present history:

- Age of onset - type of onset.
- Course and duration of the disease.
- Pattern of joint involvement
- Raynaud phenomenon and morning stiffness.
- Fever, rash, photosensitivity and other eye symptoms
- Hair falling, weight loss and (mouth and genital ulcers).

REVIEW OF THE FOLLOWING SYSTEMS.
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a] Respiratory: Cough, dyspnea and chest pain
b] Gastrointestinal; Anorexia, malaise, nausea, vomiting and abdominal distension
c] Cardiovascular: Tachycardia, palpitation, pallor and precordial pain or friction rub.
d] Urinary: Renal pain, change in the amount and colour of urine, fever.

- Functional capacity of the patients according to Steinbrocker et al. (1949) grading.

D- Past history:
- Serious disease as heart failure, renal failure or acute glomerulonephritis, chronic chest infection and chronic uveitis. - Previous trauma or operations.
- Previous admission to hospitals (date and duration)

E- History of Medication:
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- The type and duration of drugs employed in the treatment.
- Onset of steroid therapy.
- Previous intra articular corticosteroid therapy (site, date, of injections and dose)
F- Family history:
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Similar conditions in the family. consternation

II- General examination
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- Pulse, temperature and blood pressure.
- Pallor, Jaundice and mouth ulcers,
- Examination of eyes for conjunctivitis, iritis
- Examination of skin for subcutaneous nodules, rash, hair and nails.
- Examination of lymph nodes.
- Chest examination.
- Cardiovascular examination.
- Abdominal examination.
- Neurological examination:
  - Muscle power, tone reflexes and sensations.
  - Deformities.

III- Locomotor system examination

Hand examination:
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- Vascular lesions - Nails.
- Distal interphalangeal joints.
- Proximal interphalangeal joints,
- Metacarpophalangeal joints.
- Flexor tendon nodules.
Examination of the following:

- Wrist - elbow and shoulder joints
- Acromio-clavicular, sterno-clavicular and temporo-mandibular joints.
- Cervical, dorsal and lumbar spine.
- Sacroiliac, hip, knee and ankle joints.

Foot examination

- Vascular lesions - Nails
- Distal inter phalangeal joints
- Proximal inter phalangeal joints
- Metatarso -phalangeal joints
- Mid-tarsal joints.
- Subtaloid joints.
- Examination of posture and gait.

Each of the above mentioned joints will be examined according to the following:

1] Inspection:

- The overlying skin
- Muscle wasting
- Deformity.
- Swelling

2] Palpation:

- Skin temperature.
Tenderness
Swelling (site-extent and consistency)
Palpation of the bony components of the joints.

3] Movements:

- Both passive and active movements.
- Range of motion of each joint.
- Abnormal movement
- crepitus
- Protective muscle spasm.

4] Articular index scoring:

- It is a method for measurement of joint activity (inflammation), which is based on the assessment of tenderness of a series of joints. In our study we used the modified Ritchie articular index which has a great advantage of simplicity (Ritchie et al., 1968).

IV- Radiological investigations

Plain x-rays were done to the affected joints in all patients.

V- Laboratory investigations.

1- Full blood picture.
2- The Erythrocyte sedimentation rate (ESR)
3- Serum rheumatoid factor.
VI- Anthropometric landmarks and Measurements.

A] Anthropometric Landmarks

In the present study, the following plane and landmarks were used (Silverhardt, 1975).

1- The frankfort plane: It is a horizontal plane passing from the lower border of the left orbit to the upper margin of the external auditory meatus. It corresponds exactly to the plane of the visual axis of looking straight a head.

2- The vertex: It is the highest point of the top of the head in the mid sagittal plane.

3- The dactylyon: It is the tip of the middle finger.

4- The acromiale: It is the most lateral point of the acromion process.

5- The radiale: It is the external superior order of the head of the radius.

6- Stylion: The distal most point of the styloid process of the radius.

7- Iliospinale: Summit of the anterior superior iliac spine.

8- The Tibiale: It is the upper point of the inner border of the medial tibial condyle.
9- The malleolus: It is the most lower point of the internal malleolus.

10- Akropodion: The most posteriorly projecting point on the heel.

11- Pterion: The tip of the most anteriorly projecting toe.

1B] Anthropometric Measurements:

Measurements used depended on the description of Silverhardt, (1975) and Cameron, (1984).

1- Weight:

Weighing was done with the subject wearing minimal clothes and bare-footed. He/She stands in the center of the scale plate-form, then weight was recorded to the nearest 0.5 Kgm. The scale measure was periodically checked before and during the work.

2- Height:

The child was asked to stand (bare footed) straight on the foot of weighing scale with his back towards the scale. He or She was also asked to look forward so that the head lies horizontally in the Frankfort plane, which is achieved by gentle orientation applied under the mastoid process. As
the individual was standing, his heels, buttocks, upper part of the back and his head were in contact with the centimeter scale and his arms hanging at the sides. The inner tube was moved up until the end of its graduation appears or until it was locked with the middle tube. Then the middle tube was raised up and down until the head marker was tightly touched the vertex while it was parallel to the floor. The measurements were read to nearest millimeter.

3- Sitting-height:

The apparatus was placed vertically with its fixed vertical board laid down on a flat seat and the graduated were vertically in contact with a wall. The sliding board was moved up enough allowing the given child to sit down on the fixed vertical board. The child was positioned so that the head was in the Frankfort plane, the shoulders relaxed, the back straight, the upper surface of the thighs horizontal and the feet supported so that a right angle was formed with the thighs and the tendon of the biceps femoris, at the back of the knees were just clear of the table. The arms were loose at the sides and the hands rest in the child's thighs. It was essential to had the knees raised a way from the table but only to the point where a right angle was formed with the thigh to avoid any tendency to lean backwards or forwards because both situation reduce sitting -height.
Then the sliding board was moved down until it touched the vertex to measure the sitting height.

4- Span:

The subject stands against a wall abducting his two upper limbs to 90 degrees with the trunk overstretched. The elbow, wrist and finger joints were fully extended with the palm looking forward. The span was measured from the tip of the right middle finger (dactlyon) to the tip of the left middle finger, using a flexible steel tape, in centimeters.

5- Upper limb measurements:

a) Total length:

With the subject's arms and hands were fully extended by his side, the distance between the acromial and the dactlyon was measured by a steel tape.

b) Upper arm length:

The external superior border of the head of the radius (radiale) was marked, and the length from this mark to the acromiale was measured.

c) Forearm length:

It was measured from the marked head of radius to the tip of lateral styloid process.
d) Hand Measurements:

i) Hand length:

From the mid-point of a line connecting the styloid processes of the radius and ulna to the dactlyon. The hand is laid flat on a table.

ii) Hand Breadth:

From the most prominent point, outside, of the lower epiphysis of the 2nd metacarpal to the most prominent inside point of the lower epiphysis of the 5th metacarpal.

6- Lower Limb measurements:

a) Total Length:

The subject stands with the lower limbs extended and symmetrically positioned. The measurement was taken from the iliospinale to the floor.

b) Leg Length

The subject stands on a flat ground. The measurement was taken from the upper point of the inner border of the medial tibial condyle (Tibiale) to the ground.

c) Tibial length:

The measurement was taken from the tibiale to the most
lower point of the medial malleolus (The malleolus).

\[ \textbf{d- Foot measurements:} \]

\[ \textbf{i) Foot length:} \]

From the most posteriorly projecting point on the heel (akropodion) to the tip of the most anteriorly projecting toe (Pternion), when the subject is standing erect.

\[ \textbf{ii) Foot breadth:} \]

The measurement was taken from the point of the anterior epiphysis of the 1st metatarsal (the most prominent point of the inner side of the foot) and the point of the anterior epiphysis of the 5th metatarsal (the most prominent point of the outer side).
### ABBREVIATION

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANA</td>
<td>Antinuclear Antibodies.</td>
</tr>
<tr>
<td>anth.</td>
<td>anthropometric.</td>
</tr>
<tr>
<td>Br.</td>
<td>Breadth.</td>
</tr>
<tr>
<td>Chr.</td>
<td>Chronic.</td>
</tr>
<tr>
<td>chron.</td>
<td>chronological.</td>
</tr>
<tr>
<td>Fct.</td>
<td>Factor.</td>
</tr>
<tr>
<td>ht.</td>
<td>height.</td>
</tr>
<tr>
<td>irid.</td>
<td>iridocyclitis.</td>
</tr>
<tr>
<td>JRA</td>
<td>Juvenile Rheumatoid Arthritis.</td>
</tr>
<tr>
<td>leng</td>
<td>Length.</td>
</tr>
<tr>
<td>Lt.</td>
<td>Left.</td>
</tr>
<tr>
<td>NCHS</td>
<td>National Center for Health Statistics.</td>
</tr>
<tr>
<td>No.</td>
<td>Number.</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Non steroidal antinflammatory agents.</td>
</tr>
<tr>
<td>Pauc.</td>
<td>Pauciarticular.</td>
</tr>
<tr>
<td>Pol.</td>
<td>Polyarticular.</td>
</tr>
<tr>
<td>Rh</td>
<td>Rheumatoid.</td>
</tr>
<tr>
<td>Rt.</td>
<td>Right.</td>
</tr>
<tr>
<td>T.M</td>
<td>Temporomandibular.</td>
</tr>
</tbody>
</table>
Results
RESULTS

The present study comprised 40 patients attending the out patient clinic of Banha and kasr EL-Eini university hospitals. They were 27 females (67.5%) and 13 males (32.5 %), with a female to male ratio equal to 2:1. Their ages ranged between three years and one month to eighteen years (with a mean age 11.85 ± 4.38 years). They were suffering from JRA and classified according to the American Rheumatism Association Criteria (Brewer et al., 1986) into 15 patients (37.5%) with systemic onset, 8 patients (20%) with pauciarticular onset, and 17 patients (42.5%) with polyarticular onset. The duration of the disease varied between 2 months and 10 years. Twenty-six patients (76%) were receiving NSAID's and or steroid therap ≤ 4 months, and 14 patients (35%) were receiving steroid therapy > 4 months.

Thirty healthy individuals were also included as a control group, they were 19 females (63.3%) and 11 males (36.7%) with female to male ratio nearly equal to 2:1, whose ages, ranged between three years and one month to eighteen years.

On studying the control group (Table 2 and 3), we observed the following:

1- The mean weight was 36.52 ± 14.62, with a range between
17 and 65 kg. Also the weight was within the normal limits ( > 5th - < 95th centile ) when compared with the standard tables except 3 cases (10%) was above the 95th centile.

2- The mean height was 138.57 ± 17.89, with a range and between 96.5 - 166 cm. Also, the height was within the normal limits when compared with the standard tables except 3 cases (10%) was above the 95th centile and 3 cases (10%) was below the 5th centile.

On the other side, studying the disease group shows the following:

1- The mean weight was 29.01 ± 9.71 and there was statistical significance of difference between it and that of the control group, the weight ranged between 11-52 kg. Also the weight was below the 5th centile in 24 patients (60%).

2- The mean height was 131.0 ± 20.54, with a range between 79.5 - 160 cm. Also, the height was below the 5th centile in 23 patients (57.5%).

3- The growth of the female patients more affected than male especially the weight and span where the difference was statistically significance for female but not for male patients (table 4,5 and 6).
4- When we classified the patients according to their mode of onset we observed that.

[a] The mean age was 8.41 ± 3.59, 12.63 ± 3.51 and 14.5 ± 3.33 years for systemic, pauciarticular and polyarticular onset respectively, also, the mean of weight was 21.13 ± 6.05, 32.65 ± 9.81 and 34.17 kg and the mean of height was 116.9 ± 18.79, 131.81 ± 19.755 and 143.05 ± 14.36 cm. and all the above differences were highly statistical significance (table, 7).

[b] In patients with systemic onset (15 patients), both weight and height were below the 5th centile in 7 cases (46.67%) for each of them (table 8).

[c] In patients with pauciarticular onset (8 patients) both weight and height were below the 5th centile in 5 patients (62.5 %) for each of them (table 8).

[d] In patients with polyarticular onset (17 patients) the weight was below the 5th centile in 12 patients (70.59) and the height was below the 5th centile in 11 patients (64.71%) (table 8)

5- As regard to the duration of the disease, we noticed that:

[a] The span, hand and foot measurements were affected by duration above 5 years, but the difference was not statistically significant (Table 9).
[b] In patients with disease duration < 3 years (15 patients) the weight was below the 5th centile in 8 cases (53.3%) and the height was below the 5th centile in 7 cases (46.7%) (table 9).

[c] While in patients with disease duration ≥ 3 years (25 patients) both weight and height were below the 5th centile in 16 cases (64%) for each of them (Table 10).

6- When we compared the patients that received corticosteroids therapy (16 patients) and those that not received corticosteroids therapy (24 patients), we found that, all anthropometric measurements of the patients received corticosteroids were lower than that of not received corticosteroids, but this difference was not statistically significance (Table 11).

- In patients receiving NSAID's or steroid ≤ 4 months (26 patients) the weight was below the 5th centile in 15 cases (57.7%), while the height was below the 5th centile in 14 cases (53.8%) (Table 12).

- While in patients who had received steroid therapy more than 4 months (Table 12), we observed that, both weight and height were below the 5th centile in 9 cases (64.3%) for each of them.
Table {2} : Anthropometric Measurements for patients with JRA and control.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Cases</th>
<th></th>
<th>Control</th>
<th></th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1)Age</td>
<td>11.85</td>
<td>4.38</td>
<td>10.97</td>
<td>4.08</td>
<td>0.86</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>2)Duration</td>
<td>3.82</td>
<td>2.23</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3)Weight</td>
<td>29.01</td>
<td>9.71</td>
<td>36.52</td>
<td>14.62</td>
<td>2.44</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>4)Height</td>
<td>131.05</td>
<td>20.54</td>
<td>138.57</td>
<td>17.89</td>
<td>1.63</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>5)Sitting ht.</td>
<td>69.89</td>
<td>10.95</td>
<td>73.90</td>
<td>10.19</td>
<td>1.58</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>6)Span.</td>
<td>127.60</td>
<td>27.77</td>
<td>142.53</td>
<td>20.89</td>
<td>2.57</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

7)Upper Limb:

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a)Total Leng.</td>
<td>56.04</td>
<td>10.03</td>
<td>59.46</td>
<td>9.01</td>
<td>1.49</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>b)Arm Leng.</td>
<td>24.84</td>
<td>7.32</td>
<td>25.60</td>
<td>3.63</td>
<td>0.57</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>c)Forearm Leng.</td>
<td>20.18</td>
<td>4.09</td>
<td>21.13</td>
<td>3.21</td>
<td>1.09</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>d)Rt.Hand Leng.</td>
<td>16.38</td>
<td>2.21</td>
<td>16.85</td>
<td>2.60</td>
<td>0.79</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>e)Lt.Hand Leng.</td>
<td>16.12</td>
<td>2.40</td>
<td>16.86</td>
<td>2.51</td>
<td>1.25</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>f)Rt.Hand Br.</td>
<td>7.06</td>
<td>0.93</td>
<td>7.61</td>
<td>0.95</td>
<td>2.43</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>j)Lt.Hand Br.</td>
<td>7.05</td>
<td>0.90</td>
<td>7.4</td>
<td>0.92</td>
<td>1.87</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

8)Lower Limb:

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a)Total Leng.</td>
<td>75.21</td>
<td>13.84</td>
<td>80.67</td>
<td>11.84</td>
<td>1.77</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>b)Leg Leng.</td>
<td>35.66</td>
<td>6.05</td>
<td>37.68</td>
<td>5.66</td>
<td>1.43</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>c)Tibial Leng.</td>
<td>31.00</td>
<td>5.52</td>
<td>32.35</td>
<td>5.05</td>
<td>1.07</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>d)Rt.Foot Leng.</td>
<td>20.71</td>
<td>3.28</td>
<td>21.83</td>
<td>2.66</td>
<td>1.56</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>e)Lt.Foot Leng.</td>
<td>20.57</td>
<td>3.13</td>
<td>22.02</td>
<td>2.59</td>
<td>2.11</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>f)Rt.Foot Br.</td>
<td>8.23</td>
<td>1.29</td>
<td>9.09</td>
<td>2.51</td>
<td>1.72</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>j)Lt.Foot Br.</td>
<td>8.21</td>
<td>1.225</td>
<td>8.75</td>
<td>1.16</td>
<td>1.87</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant
P < 0.05 = significant

This table shows that all the anthropometric measurements of the patients are below that of the control and these differences are statistically significant for weight, span and breadth of the right hand.
Table (3): Centiles of the weight and height of the patients with JRA and control groups.

<table>
<thead>
<tr>
<th>groups</th>
<th>Weight</th>
<th></th>
<th>Height</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5th</td>
<td>5th-95th</td>
<td>&gt;95th</td>
<td>&lt;5th</td>
<td>5th-95th</td>
<td>&gt;95th</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Patients</td>
<td>24</td>
<td>60%</td>
<td>16</td>
<td>40%</td>
<td>0</td>
<td>0%</td>
<td>23</td>
<td>37.5%</td>
</tr>
<tr>
<td>No=40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>control</td>
<td>0</td>
<td>0%</td>
<td>27</td>
<td>90%</td>
<td>3</td>
<td>10%</td>
<td>3</td>
<td>10%</td>
</tr>
<tr>
<td>No=30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This table shows that the weight is below the 5th centile in 60% of the patients, compared to 0% of the control. Also the height was below the 5th centile in 37.5% of the patients compared to 10% of the control.
Table 4: Anthropometric Measurements for male patients and control

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Cases</th>
<th>Control</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td>T</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>1) Age</td>
<td>11.3</td>
<td>4.00</td>
<td>10.41</td>
<td>4.42</td>
<td>0.59</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>2) Duration</td>
<td>4.21</td>
<td>2.34</td>
<td>----</td>
<td>----</td>
<td>----</td>
<td>----</td>
<td></td>
</tr>
<tr>
<td>2) Weight</td>
<td>28.31</td>
<td>6.81</td>
<td>34.86</td>
<td>15.31</td>
<td>0.31</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>4) Height</td>
<td>132.23</td>
<td>17.44</td>
<td>136.5</td>
<td>17.82</td>
<td>0.59</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>3) Sitting ht.</td>
<td>69.92</td>
<td>31.12</td>
<td>71.73</td>
<td>11.11</td>
<td>0.36</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>6) Span.</td>
<td>133.35</td>
<td>18.50</td>
<td>139.559</td>
<td>20.60</td>
<td>0.77</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>7) Upper Limb:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>56.5</td>
<td>9.17</td>
<td>58.52</td>
<td>9.662</td>
<td>0.52</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>b) Arm Leng.</td>
<td>24.17</td>
<td>3.99</td>
<td>24.69</td>
<td>3.63</td>
<td>0.34</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>c) Forearm Leng.</td>
<td>20.67</td>
<td>4.10</td>
<td>20.85</td>
<td>3.38</td>
<td>0.12</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>d) Rt. Hand Leng.</td>
<td>16.49</td>
<td>1.49</td>
<td>17.05</td>
<td>2.67</td>
<td>0.57</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>e) Lt. Hand Leng.</td>
<td>16.53</td>
<td>2.03</td>
<td>16.97</td>
<td>2.68</td>
<td>0.45</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>f) Rt. Hand Br.</td>
<td>7.19</td>
<td>0.64</td>
<td>7.66</td>
<td>1.02</td>
<td>1.32</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>j) Lt. Hand Br.</td>
<td>7.06</td>
<td>0.60</td>
<td>7.52</td>
<td>0.99</td>
<td>1.33</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>8) Lower Limb:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>74.9</td>
<td>14.36</td>
<td>78.86</td>
<td>10.69</td>
<td>0.81</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>b) Leg Leng.</td>
<td>35.15</td>
<td>55.10</td>
<td>36.77</td>
<td>5.96</td>
<td>0.69</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>c) Tibial Leng.</td>
<td>30.31</td>
<td>4.77</td>
<td>31.45</td>
<td>5.49</td>
<td>0.54</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>d) Rt. Foot Leng.</td>
<td>21.05</td>
<td>2.67</td>
<td>22.04</td>
<td>2.81</td>
<td>0.87</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>e) Lt. Foot Leng.</td>
<td>20.85</td>
<td>2.55</td>
<td>21.97</td>
<td>2.77</td>
<td>1.03</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>f) Rt. Foot Br.</td>
<td>8.40</td>
<td>1.02</td>
<td>8.52</td>
<td>1.45</td>
<td>0.4</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>j) Lt. Foot Br.</td>
<td>8.22</td>
<td>1.15</td>
<td>8.67</td>
<td>1.36</td>
<td>0.86</td>
<td>&gt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant
P < 0.05 = significant

This table shows that the anth. parameters of the male patients are below that of the control one, but there is no statistical significant of difference.
HEIGHT AND WEIGHT FOR MALE PATIENT, CONTROL AND STANDARD

Fig. (1)
Table 5: Anthropometric Measurements for female patients and control

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Cases Mean</th>
<th>Cases S.D.</th>
<th>Control Mean</th>
<th>Control S.D.</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Age</td>
<td>12.11</td>
<td>4.60</td>
<td>11.29</td>
<td>4.48</td>
<td>0.61</td>
<td>&gt; .05</td>
</tr>
<tr>
<td>2) Duration</td>
<td>3.68</td>
<td>2.17</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3) Weight</td>
<td>29.35</td>
<td>10.94</td>
<td>37.47</td>
<td>14.54</td>
<td>0.46</td>
<td>&lt; .05</td>
</tr>
<tr>
<td>4) Height</td>
<td>130.48</td>
<td>22.17</td>
<td>139.76</td>
<td>18.97</td>
<td>0.48</td>
<td>&gt; .05</td>
</tr>
<tr>
<td>5) Sitting ht.</td>
<td>69.87</td>
<td>31.21</td>
<td>75.16</td>
<td>9.71</td>
<td>0.85</td>
<td>&gt; .05</td>
</tr>
<tr>
<td>6) Span.</td>
<td>124.83</td>
<td>4.15</td>
<td>144.23</td>
<td>21.41</td>
<td>0.59</td>
<td>&lt; .05</td>
</tr>
</tbody>
</table>

7) Upper Limb:

- Total Leng. 55.82 2.20 60.00 8.87 0.42 > 0.05
- Arm Leng. 25.17 10.59 26.13 4.61 1.05 > 0.05
- Forearm Leng. 19.95 8.52 21.3 3.20 0.366 > 0.05
- Right Hand Leng. 16.33 2.37 16.73 2.62 0.53 > 0.05
- Left Hand Leng. 15.92 2.57 16.8 2.48 1.17 > 0.05
- Right Hand Br. 6.99 1.04 7.58 0.93 0.23 > 0.05
- Left Hand Br. 7.04 1.03 7.43 0.90 0.25 > 0.05

8) Lower Limb:

- Total Leng. 75.46 10.01 81.71 12.77 0.65 > 0.05
- Leg Leng. 35.91 13.85 38.21 5.57 0.65 > 0.05
- Tibial Leng. 31.33 6.38 32.87 4.86 0.71 > 0.05
- Right Foot Leng. 20.54 3.58 21.71 2.64 1.27 > 0.05
- Left Foot Leng. 20.44 3.42 22.05 2.56 1.82 > 0.05
- Right Foot Br. 0.19 1.42 9.42 2.94 1.69 > 0.05
- Left Foot Br. 8.20 1.31 8.79 1.06 1.69 > 0.05

P > 0.05 = Not significant
P < 0.05 = significant

This table shows that the anthropometric parameters of the female patients are below that of the control and these differences are statistically significant for weight and span.
Table {6} : Anthropometric Measurements for male and female patients

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>1) Age</td>
<td>11.30</td>
<td>4.00</td>
</tr>
<tr>
<td>2) Duration</td>
<td>4.12</td>
<td>2.34</td>
</tr>
<tr>
<td>3) Weight</td>
<td>28.85</td>
<td>6.81</td>
</tr>
<tr>
<td>4) Height</td>
<td>132.46</td>
<td>17.44</td>
</tr>
<tr>
<td>5) Sitting ht.</td>
<td>69.92</td>
<td>3.12</td>
</tr>
<tr>
<td>6) Span.</td>
<td>133.35</td>
<td>18.50</td>
</tr>
<tr>
<td>7) Upper Limb:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>56.4</td>
<td>9.17</td>
</tr>
<tr>
<td>b) Arm Leng.</td>
<td>24.17</td>
<td>3.99</td>
</tr>
<tr>
<td>c) Forearm Leng.</td>
<td>20.67</td>
<td>4.10</td>
</tr>
<tr>
<td>d) Rt. Hand Leng.</td>
<td>16.49</td>
<td>1.93</td>
</tr>
<tr>
<td>e) Lt. Hand Leng.</td>
<td>16.53</td>
<td>2.03</td>
</tr>
<tr>
<td>f) Rt. Hand Br.</td>
<td>7.19</td>
<td>0.64</td>
</tr>
<tr>
<td>j) Lt. Hand Br.</td>
<td>7.06</td>
<td>0.60</td>
</tr>
<tr>
<td>8) Lower Limb:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>74.69</td>
<td>14.36</td>
</tr>
<tr>
<td>b) Leg Leng.</td>
<td>35.15</td>
<td>5.51</td>
</tr>
<tr>
<td>c) Tibial Leng.</td>
<td>30.31</td>
<td>4.77</td>
</tr>
<tr>
<td>d) Rt. Foot Leng.</td>
<td>21.05</td>
<td>2.67</td>
</tr>
<tr>
<td>e) Lt. Foot Leng.</td>
<td>20.85</td>
<td>2.55</td>
</tr>
<tr>
<td>f) Rt. Foot Br.</td>
<td>8.30</td>
<td>1.02</td>
</tr>
<tr>
<td>j) Lt. Foot Br.</td>
<td>0.22</td>
<td>1.15</td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant  
P < 0.05 = Significant

This table shows that the anthropometric parameters of the female patients are below that of the males except for the lower limb measurements, but these differences not reach any statistical significance.
Table {7} : Anthropometric Measurements for all patients with JRA according to the type of onset

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Systemic No. = 15</th>
<th>Pauc. No.=8</th>
<th>Pol. No.=17</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>1) Age</td>
<td>8.41</td>
<td>3.59</td>
<td>12.63</td>
<td>3.51</td>
<td>14.50</td>
</tr>
<tr>
<td>2) Duration</td>
<td>3.66</td>
<td>2.12</td>
<td>4.25</td>
<td>2.76</td>
<td>3.76</td>
</tr>
<tr>
<td>3) Weight</td>
<td>21.13</td>
<td>6.05</td>
<td>32.56</td>
<td>9.81</td>
<td>34.17</td>
</tr>
<tr>
<td>4) Height</td>
<td>116.90</td>
<td>18.79</td>
<td>131.81</td>
<td>19.75</td>
<td>143.05</td>
</tr>
<tr>
<td>5) Sitting ht.</td>
<td>63.63</td>
<td>8.24</td>
<td>66.68</td>
<td>14.01</td>
<td>76.79</td>
</tr>
<tr>
<td>6) Span.</td>
<td>114.76</td>
<td>19.54</td>
<td>120.56</td>
<td>42.63</td>
<td>142.11</td>
</tr>
</tbody>
</table>

7) Upper Limb:

<table>
<thead>
<tr>
<th>Measurements</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Total Leng.</td>
<td>48.32</td>
</tr>
<tr>
<td>b) Arm Leng.</td>
<td>20.80</td>
</tr>
<tr>
<td>c) Forearm Leng.</td>
<td>17.12</td>
</tr>
<tr>
<td>d) Rt. Hand Leng.</td>
<td>14.46</td>
</tr>
<tr>
<td>e) Lt. Hand Leng.</td>
<td>14.50</td>
</tr>
<tr>
<td>f) Rt. Hand Br.</td>
<td>6.37</td>
</tr>
<tr>
<td>j) Lt. Hand Br.</td>
<td>6.51</td>
</tr>
</tbody>
</table>

8) Lower Limb:

<table>
<thead>
<tr>
<th>Measurements</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Total Leng.</td>
<td>64.1</td>
</tr>
<tr>
<td>b) Leg Leng.</td>
<td>31.06</td>
</tr>
<tr>
<td>c) Tibial Leng.</td>
<td>26.73</td>
</tr>
<tr>
<td>d) Rt. Foot Leng.</td>
<td>17.97</td>
</tr>
<tr>
<td>e) Lt. Foot Leng.</td>
<td>18.15</td>
</tr>
<tr>
<td>f) Rt. Foot Br.</td>
<td>7.28</td>
</tr>
<tr>
<td>j) Lt. Foot Br.</td>
<td>7.30</td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant
P < 0.05 = significant
P < 0.001 = highly significant.

This table shows that the mean age and all anthropometric measurements for patients with systemic onset are below that of pauciarticular, below that of polyarticular except for arm and forearm length where the differences are highly statistical significant.
HEIGHT AND WEIGHT FOR CASES
ACCORDING TO ONSET OF DISEASE

Fig. (3)
Table (8): Centiles of the weight and height of patients according to mode of onset.

<table>
<thead>
<tr>
<th>Type of onset</th>
<th>Weight</th>
<th>Height</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5th</td>
<td>&gt;5th-&lt;95th</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>1-Systemic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>onset</td>
<td>7</td>
<td>46.67%</td>
</tr>
<tr>
<td>No= 15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-Pauciarticular</td>
<td>5</td>
<td>62.5%</td>
</tr>
<tr>
<td>Onset No=8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-Polyarticular</td>
<td>12</td>
<td>70.59%</td>
</tr>
<tr>
<td>onset No=17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This table shows that the percentage of weight and height which are below 5th centile are larger in polyarticular onset than other 2 types.
Table 9: Anthropometric Measurements for patients with JRA according to the duration of the disease.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>&lt;3</th>
<th>3-&lt;5</th>
<th>≥5</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>1) Age</td>
<td>10.68</td>
<td>4.58</td>
<td>11.37</td>
<td>4.19</td>
<td>13.90</td>
</tr>
<tr>
<td>2) Duration</td>
<td>1.8</td>
<td>0.56</td>
<td>3.37</td>
<td>0.53</td>
<td>6.86</td>
</tr>
<tr>
<td>3) Weight</td>
<td>26.76</td>
<td>10.68</td>
<td>28.34</td>
<td>10.01</td>
<td>32.45</td>
</tr>
<tr>
<td>4) Height</td>
<td>129.69</td>
<td>23.79</td>
<td>130.15</td>
<td>22.19</td>
<td>133.63</td>
</tr>
<tr>
<td>5) Sitting ht.</td>
<td>67.11</td>
<td>13.98</td>
<td>70.87</td>
<td>9.81</td>
<td>71.54</td>
</tr>
<tr>
<td>6) Span</td>
<td>128.84</td>
<td>27.07</td>
<td>131.68</td>
<td>21.15</td>
<td>120.36</td>
</tr>
</tbody>
</table>

7) Upper Limb:

<table>
<thead>
<tr>
<th>Part</th>
<th>Leng.</th>
<th>Mean</th>
<th>S.D.</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Total</td>
<td>55.28</td>
<td>11.41</td>
<td>56.25</td>
<td>10.80</td>
<td>56.60</td>
</tr>
<tr>
<td>b) Arm</td>
<td>23.69</td>
<td>5.54</td>
<td>23.98</td>
<td>4.07</td>
<td>27.45</td>
</tr>
<tr>
<td>c) Forearm</td>
<td>19.34</td>
<td>3.98</td>
<td>20.11</td>
<td>4.04</td>
<td>21.24</td>
</tr>
<tr>
<td>d) Rt. Hand</td>
<td>15.96</td>
<td>2.63</td>
<td>16.62</td>
<td>2.24</td>
<td>16.46</td>
</tr>
<tr>
<td>e) Lt. Hand</td>
<td>15.92</td>
<td>2.74</td>
<td>16.25</td>
<td>2.52</td>
<td>16.08</td>
</tr>
<tr>
<td>f) Rt. Hand Br.</td>
<td>6.85</td>
<td>1.13</td>
<td>7.16</td>
<td>0.96</td>
<td>7.10</td>
</tr>
<tr>
<td>j) Lt. Hand Br.</td>
<td>6.90</td>
<td>1.10</td>
<td>7.23</td>
<td>0.91</td>
<td>7.00</td>
</tr>
</tbody>
</table>

8) Lower Limb:

<table>
<thead>
<tr>
<th>Part</th>
<th>Leng.</th>
<th>Mean</th>
<th>S.D.</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Total</td>
<td>73.80</td>
<td>16.40</td>
<td>74.12</td>
<td>15.54</td>
<td>78.31</td>
</tr>
<tr>
<td>b) Leg</td>
<td>34.88</td>
<td>7.61</td>
<td>35.37</td>
<td>6.28</td>
<td>36.90</td>
</tr>
<tr>
<td>c) Tibial</td>
<td>30</td>
<td>7.13</td>
<td>30.71</td>
<td>5.59</td>
<td>32.04</td>
</tr>
<tr>
<td>d) Rt. Foot</td>
<td>20.19</td>
<td>3.78</td>
<td>20.91</td>
<td>3.35</td>
<td>20.97</td>
</tr>
<tr>
<td>e) Lt. Foot</td>
<td>19.97</td>
<td>3.97</td>
<td>20.95</td>
<td>3.44</td>
<td>20.68</td>
</tr>
<tr>
<td>f) Rt. Foot Br.</td>
<td>7.82</td>
<td>1.51</td>
<td>8.43</td>
<td>1.36</td>
<td>8.36</td>
</tr>
<tr>
<td>j) Lt. Foot Br.</td>
<td>7.82</td>
<td>1.38</td>
<td>8.7</td>
<td>1.34</td>
<td>8.4</td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant
P < 0.05 = Significant

This table shows that the anthropometric measurements are affected by duration above 5 years for span, hand and foot measurements and not for other anthropometric measurements, and these differences not reach statistical significance.
HEIGHT AND WEIGHT FOR CASES ACCORDING TO DISEASE DURATION

Fig. (4)
Table (10): Centiles of weight and height of patients with JRA according to the duration of the disease.

<table>
<thead>
<tr>
<th>Type of onset</th>
<th>weight</th>
<th>Height</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5th</td>
<td>&gt;5th-&lt;95th</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>&lt; 3 years</td>
<td>8</td>
<td>53.3</td>
</tr>
<tr>
<td>No = 15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 3 years</td>
<td>16</td>
<td>64%</td>
</tr>
<tr>
<td>No = 25</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This table shows that, the percentage of weight and height which are below 5th centile increases with the duration of the disease.
Table 11: Anthropometric Measurements for patients that received steroid and that not received steroid therapy.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Steroid</th>
<th>Non steroid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 16</td>
<td>N = 24</td>
</tr>
<tr>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>1) Age</td>
<td>11.24</td>
<td>4.30</td>
</tr>
<tr>
<td>2) Duration</td>
<td>5.03</td>
<td>2.60</td>
</tr>
<tr>
<td>3) Weight</td>
<td>27.16</td>
<td>7.94</td>
</tr>
<tr>
<td>4) Height</td>
<td>125.44</td>
<td>19.61</td>
</tr>
<tr>
<td>5) Sitting ht.</td>
<td>68.38</td>
<td>9.57</td>
</tr>
<tr>
<td>6) Span.</td>
<td>118.13</td>
<td>33.53</td>
</tr>
<tr>
<td>7) Upper Limb:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>52.94</td>
<td>9.86</td>
</tr>
<tr>
<td>b) Arm Leng.</td>
<td>24.44</td>
<td>10.45</td>
</tr>
<tr>
<td>c) Forearm Leng.</td>
<td>19.48</td>
<td>4.74</td>
</tr>
<tr>
<td>d) R. Hand Leng.</td>
<td>15.7</td>
<td>2.00</td>
</tr>
<tr>
<td>e) Lt. Hand Leng.</td>
<td>15.46</td>
<td>2.43</td>
</tr>
<tr>
<td>f) R. Hand Br.</td>
<td>6.73</td>
<td>0.89</td>
</tr>
<tr>
<td>g) Lt. Hand Br.</td>
<td>6.83</td>
<td>0.83</td>
</tr>
<tr>
<td>8) Lower Limb:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Total Leng.</td>
<td>70.56</td>
<td>14.14</td>
</tr>
<tr>
<td>b) Leg Leng.</td>
<td>34.25</td>
<td>6.14</td>
</tr>
<tr>
<td>c) Tibial Leng.</td>
<td>29.78</td>
<td>5.41</td>
</tr>
<tr>
<td>d) R. Foot Leng.</td>
<td>19.49</td>
<td>3.37</td>
</tr>
<tr>
<td>e) Lt. Foot Leng.</td>
<td>19.54</td>
<td>3.21</td>
</tr>
<tr>
<td>f) R. Foot Br.</td>
<td>7.78</td>
<td>1.25</td>
</tr>
<tr>
<td>g) Lt. Foot Br.</td>
<td>7.87</td>
<td>1.17</td>
</tr>
</tbody>
</table>

P > 0.05 = Not significant  
P < 0.05 = significant

This table shows that the anthropometric measurements of patients that received corticosteroids therapy are lower than that not received corticosteroids therapy, but this difference not reach statistical significance.
HEIGHT & WEIGHT FOR MALE PATIENTS
WITH & WITHOUT STEROID THERAPY

Fig. (5)
HEIGHT & WEIGHT FOR FEMALE PATIENTS
WITH & WITHOUT STEROID THERAPY

Fig. (6)
Table (12): Centiles of weight and height of the patients according to the mode of therapy.

<table>
<thead>
<tr>
<th>Type of onset</th>
<th>weight</th>
<th>Height</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5th</td>
<td>&gt;5th-&lt;95th</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>NSAID's or steroid &lt; 4 Mo.</td>
<td>15</td>
<td>57.7</td>
</tr>
<tr>
<td>No =26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steroid ≥ 4 months</td>
<td>9</td>
<td>64.3</td>
</tr>
<tr>
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This table shows that, the percentile of weight and height that are below the 5th centile are larger in patients with steroid therapy ≥ 4 months more than that received NSAID'S or steroid < 4 months.
DISCUSSION

Anthropometric measurements have certain characteristics that distinguish them from other types of tests used in the assessment of growth and development. They are very closely related to the age of the subject and have the unique quality of being a measure of size (McLaren and Burman, 1982).

Because body size, shape and body composition are functions of growth and development, no single physical indicator can give a complete assessment of the process. Therefore, multiple indicators must be used (Vermeersch et al., 1984).

In JRA, growth disturbances adjacent to the inflamed joints may result in either overgrowth or undergrowth of the affected part. Increased leg length may follow chronic arthritis of the knee, and micrognathia after temporomandibular arthritis may be a late hallmark of JRA. Small, deformed feet may result from foot involvement in early childhood and shortened fingers from early hand involvement (Nelson, et al., 1992).

The aim of this study is to identify the patterns of growth among children with JRA particularly the segmental
growth. Also, to compare the growth of the patients group with that of control group and the standard (NCHS).

In our study the number of female patients was 27 (67.5\%) and the number of male patients was 13 (32.5\%). So, the female in our study accounted for the majority of cases and the ratio of females to males was about (or more than) 2:1.

This finding was in agreement with EL-Awar (1987) and Cassidy (1989) who stated that, girls account for the majority of cases in the early peak of the distribution curve and are affected over all at least twice as often as boys.

In our study, we found that the anthropometric measurements of the patients studied were below that of the control and the difference was statistically significant for, weight, span and breadth of the right hand.

Also, we observed that the weight of 24 patients (60\% of all patients) was below the 5th centile, while the weight of the control group was within normal limits compared with the standard tables, even above 95th centile in 3 subjects (10\%) of the control group.
The results was in agreement with the results of EL Awar (1987), who found that the weight of the patients with JRA was below the 3rd centile in 23.33% of cases. Also, in agreement with the suggestion of Miller et al. (1960) that the weight loss is a feature of any chronic inflammatory diseases.

This could be explained by the fact that, the weight is the best index of nutrition and growth, also growth responses are observed in weight before they are noted in other aspects of growth (Kempe and Silver, 1980).

In the present study, we observed that the mean height of the patients (131.05 ± 20.54) was lower than that of the control group (138.57 ±17.89) but the difference did not reach statistical significance. Also the height of the patients was below the 5th centile in 23 patients (57.5%) while it was below the 5th centile in only 3 subjects (10%) and above the 95th centile in another 3 subjects (10%) of the control group.

This finding was in agreement with , Bernstein et al., (1977), Cassidy (1989) and Davies (1989), who suggested that, linear growth is usually retarded and stunting of growth may occur from early closure of the epiphyses in patients with JRA.
Also, our results coincide with the finding of EL Awar (1987), who found that the height of the patients was below the 3rd centile in 12 patients (40%), while it was below the 3rd centile in 2 subjects (6.66%) of the control group.

From the above, we observed that retardation of growth occurred in more than half of all patients with JRA.

This observation go hand in hand with the suggestion of Ansell and Bywaters (1956), that the impairment of general growth and development occurs in half of all JRA patients, which is related to active disease as well as to prolonged corticosteroids administration.

Statistical significance of difference was found between the diseased group and the control as regard to span and breadth of the right hand. This may be due to growth disturbances adjacent to inflamed joints in the upper limb resulting in undergrowth of the affected part especially the right hand and also due to shortened fingers from early hand involvement.

This suggestion coincide with Nelson et al. (1992), who stated that, growth disturbances adjacent to inflamed joints
may result in either overgrowth or undergrowth of the affected part.

Also, in agreement with Cassidy (1989) who stated that, brachydactyly develops in patients with JRA from premature closure of the epiphyseal growth plates.

Or may be due to deformities in the upper limbs that may accompany JRA.

Cassidy (1989) emphasized that, secondary growth deformities are frequent in children with limited joint disease. This may be due to or aggravated by corticosteroids therapy, and this suggestion coincide with Tomminga et al., (1992) who observed that, the retardation of arm span was significantly larger than the retardation of sitting height in children with acute lymphoblastic leukemia receiving corticosteroids therapy. They concluded that, corticosteroids medication in the most acceptable explanation for their finding.

In our results we found that in the male patients all the anthropometric measurements taken were below that of the control group but these differences did not reach statistical significance, while in the female patients these
differences were statistically significant for weight and span.

This finding could be explained by the statement of Tanner et al., (1975), that in some populations boys are more protected from adverse environmental circumstances than are girls.

In the present study, according to the mode of the disease onset, a highly significant differences was observed between all anthropometric parameters in patients with systemic onset and those with pauciarticular and polyarticular onset except for arm and forearm length, where that for polyarticular were below that of pauciarticular onset.

This differences may be attributed to the differences in the chronological age between the three types of onset, but as regard to the arm and forearm length, where that for polyarticular were below that of pauciarticular onset, although the mean age of polyarticular onset were higher than that of pauciarticular onset, may be due to that the polyarticular onset affected the growth more than other 2 types, especially the arm and forearm length.

This is supported by the following finding that, each
of the weight and height were below 5th centile in 46.67 % of patients with systemic onset and 62.5 % of patients with pauciarticular onset but in polyarticular onset patients, the weight was below the 5th centile in 70.59 % and the height was below the 5th centile in 64.71 %.

This in agreement with Ansell (1978), who found that in a twin study allowed to look at growth that the one who had polyarticular onset was treated with prednisolone for almost 7 years is considerably smaller than her fit monozygotic twin.

In the present study we observed that the anthropometric measurements were affected by a disease duration above 5 years for span, hand and foot measurements but the difference did not reach statistical significance. Also we noticed that the weight of the patients with a disease duration < 3 years was below the 5th centile in 8 cases (53.3 %) and the height was below the 5th centile in 7 cases (46.7%) while in patients with a disease duration ≥ 3 years, both weight and height were below the 5th centile in 16 cases (64%) for each of them.

So, growth of the patients represented by weight, height, span, hand and foot measurements correlated with
the duration of the disease, but the difference did not reach statistical significance, which could be explained by the difference in the chronological age between the three groups in the direction against the duration, where we found that, the mean age for group duration ≤ 3 years was 10.68 years and for group duration 3 - 5 years was 11.37, while that for group duration > 5 years was 13.90 years, and this was evidenced in weight and height when we compared the percentiles, where we found that the weight was below the 5th centile in 53.3% of patients with disease duration < 3 years and the height was below the 5th centile in 46.7% while both weight and height were below the 5th centile in 64% with disease duration ≥ 3 years.

Or may be due to small number of the patients in each group, also many factors may interact rather than the duration of the disease alone e.g. type of onset, age of onset, course of the disease, periods of disease activity and mode of therapy.

This finding was in agreement with the finding of El Awar (1987) who found that, there were no statistical significance of difference for weight and height as regard to the duration of the disease.
In the present study, we found that the anthropometric measurements of patients who received corticosteroids therapy were below that who did not received corticosteroids therapy, but this difference did not reach statistical significance.

Also, we observed that, in patients on NSAID’s or steroids for less than 4 months duration, the weight was below the 5th centile in 15 cases (57.7%) and the height was below the 5th centile in 14 cases (53.8%), while in patients on steroid for more than 4 months duration the weight and height were below the 5th centile in 9 cases (64.3%) for both of them.

From the above, it was evident that, corticosteroids therapy had suppressed the growth of patients with JRA and this was in agreement with Byron et al (1983) and Davies (1989) who stated that not only does juvenile chronic arthritis appear to impair linear growth in children, but that growth can be further compromised by corticosteroids.

In our results there was no significant statistical difference between all anthropometric measurements in patients who did not received corticosteroids therapy and those received corticosteroids therapy, and this was in agreement with the result of EL-Awar (1987).
This result does not contradict the well-known fact that steroid therapy plays an important role in growth retardation in children, but may be due to, the small number of patients in each group. Not only that, but most of our patients in the steroid therapy group had received more than one steroid regimen either simultaneously or at different periods along the disease course.

From all the above, we can concluded that, growth retardation occurred in patients with JRA in all growth parameters and some of these parameters reach statistical significance of difference especially in female patients.

SUMMARY AND CONCLUSION

Disturbances of growth are characteristic features of arthritis in children,

Anthropometry appears to be of greatest value as it detects deviations from the usual pattern characteristic of the growth period.

This work aimed to identify the patterns of growth among children with JRA particularly the segmental growth, and to compare the growth of the patients group with that of the control group and the standard.

To achieve this aim, a cross sectional study was carried out upon 40 patients with JRA (27 females and 13 males) ranged in age between three years and one month to eighteen years with a mean age of 11.85 years and 30 healthy individuals as a control group (19 females and 11 males) ranged in age between three years and one month to eighteen years with mean age of 10.97 years attending the out patient clinic of Banha and kasr EL-Eini university hospitals. Each child in studied groups was subjected to the following examination:

- All patients were subjected to full history taking, thorough clinical examination, and routine investigations to
confirm the diagnosis according to the American Rheumatism Association criteria.

- The control group were subjected to full history taking and thorough clinical examination to exclude any individual suffering from any chronic disease.

- Assessment of growth through measurements of the following parameters: Weight, height, sitting height, span, upper limb measurements (total length, upper arm length, forearm length, hand length and breadth) and lower limb measurements (total length, leg length tibial length and foot length and breadth).

- It was observed that the mean weight in the patients group was 29.01 ± 9.71 Kg., while it was 36.52 ± 14.62 in the control group and there was statistical significance of difference between them. The range of the weight of the patients was 11-52kg., while it was 17-65 kg. in the control group. Also, the weight of the patients was below the 5th centile in 24 patients (60%), while that of the control group was within normal limits (>5th - 95th centile) even above the normal limits (95th centile) in 3 cases (10%) and no any one of the control group lies below the 5th centile (0%).

While, as regard to the height, we noticed that the mean height of the patients was 131.0 ± 20.54, while it was
138.57 ± 17.8, and the difference did not reach statistical significance. Also, the range of the height of the patients was 79.5-160 cm., while it was 96.5-166 cm. in the control group. We observed that, the height of the patients was below the 5th centile in 23 patients (57.5%), while that of the control group was within the normal limits (5th - 95th centile) except 3 individuals (10%) was above the 95th centile.

It was observed that, the growth of the female patients more affected than male patients especially the weight and span, where the difference was statistically significance for female patients but not for male patients.

When we classified the patients according to their mode of onset we observed that:

- The mean age was 8.41 ± 3.59, 12.63 ± 3.51 and 14.5 ± 3.33 years for systemic, pauciarticular and polyarticular onset respectively, also, the mean of weight was 21.13 ± 6.05, 32.65 ± 9.81 and 34.17 kg and the mean of height was 116.9 ± 18.79, 131.81 ± 19.755 and 143.05 ± 14.36 cm. and all the above differences were highly statistical significance.

- In patients with systemic onset (15 patients), both weight and height were below the 5th centile in 7 cases (46.67%) for each of them.
In patients with pauciarticular onset (8 patients) both weight and height were below the 5th centile in 5 patients (62.5%) for each of them.

In patients with polyarticular onset (17 patients) the weight was below the 5th centile in 12 patients (70.59) and the height was below the 5th centile in 11 patients (64.71%).

As regard to the duration of the disease, we noticed that:

The span, hand and foot measurements were affected by duration above 5 years, but the difference was not statistically significant.

In patients with disease duration < 3 years (15 patients) the weight was below the 5th centile in 8 cases (53.3%) and the height was below the 5th centile in 7 cases (46.7%).

While in patients with disease duration ≥ 3 years (25 patients) both weight and height were below the 5th centile in 16 cases (64%) for each of them.

When we compared the patients that received corticosteroids therapy (16 patients) and those that not received corticosteroids therapy (24 patients), we found
that, all anthropometric measurements of the patients received corticosteroids were lower than that of not received corticosteroids, but this difference was not statistically significance.

- In patients receiving NSAID's or steroid ≤ 4 months (26 patients) the weight was below the 5th centile in 15 cases (57.7 %), while the height was below the 5th centile in 14 cases (53.8%)

- While in patients who had received steroid therapy more than 4 months (14 patients) we observed that, both weight and height were below the 5th centile in 9 cases (64.3 %) for each of them.

Lastly, we can concluded that, growth retardation occurred in patients with JRA in all growths parameters and some of these parameters reaches statistical significance of difference (such as weight, span and breadth of the right hand) especially in female patients.

Also, the impairment of growth increases with increasing the duration of the disease and further compromised by corticosteroid administration.

Also, we recommended a prospective large scaled studies on different growth parameters to clarify the issue of any significant difference between different variants.
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الخلاصة العربية

الاختلافات في النمو ظاهرة خاصة للالتهابات المفصلية في الأطفال، وللبقاعات البشرية اهمية كبيرة في تحديد انحرافات نمو الأطفال الطبيعي المميز لمراحل النمو.

هذا العمل يهدف إلى توضيح انماط النمو خلال الأطفال المصابون بمرض الروماتويد الحدثي خاصة النمو المقطعي ومقارنة النمو في المجموعة المرفقية بالقرن تحديعة ومعيار العالمي (الجدول القياسي).

لتحقيق هذه الهدف أجريت دراسة مقطعيه على 40 مريض (14 نان + 16 ذكور) مصابون بمرض الروماتويد الحدثي ويتراوح عمرهم بين 3 سنوات وشهر يلي 18 سنة مع متوسط عمر 11,85 سنة بالإضافة إلى عينة تحديعة من الاشخاص الطبيعيين 30 شخصا (19 نان + 11 ذكور) وتتراوح عمرهم بين 3 سنوات وشهر يلي 18 سنة مع متوسط عمر 10,97 سنة يترددون على العيادات الخارجية بالمستشفيات الجامعية بينهما والأقصر العيني وقد خضع كل طفل في المجاعات التي أجريت عليها الدراسة للفحوصات الأدائية:

1) بالنسبة لكل المرضى قد أخذ منهم تاريخ كامل عن المرض وقد خضعوا للفحوصات الإكلينيكية والوسائل المعملية للتأكد من التشخيص حسب معايير الجمعية الأمريكية للروماتيزم.

2) بالنسبة للمجموعة البحضية فقد أخذ منهم تاريخ مرض وقد خضعوا للفحوصات الإكلينيكية استبعادياً شعبياً منهم يعاني من أي مرض مزمن تقييم النمو البدني من خلال المقاسات الإندرومبترية الادائية: الوزن والطول وارتفاع الجذع وطول الباطن ومقاسات الطرف العلوي (طول الطرف العلوي الكلي وطول الذراع العلوي وطول الساعد وطول اليد وعرض اليد) ومقاسات الطرف السفلي (طول الطرف السفلي الكلي وطول الساق وطول العيني وطول القدم وعرض القدم).
وقد لوحظ أن متوسط الوزن في المجموعة المرفعة كان 29,01 ± 9,71 كجم بينما كان 36,06 ± 14,13 كجم في المجموعة التحكمية. وكان لهذا الاختلاف دلالة إحصائية.

كانت الوزن تتراوح بين 11-22 كجم في المجموعة المرفعة بينما كان يتراوح بين 17-65 كجم في المجموعة التحكمية.

وايضاً كان الوزن أقل من المعدل الخامس في 44 مريض (44%) في حين انه كان في الحدود الطبيعية (حتى أنه كان فوق المعدل الطبيعي) في 39 مريض (39%) والبعض في ثلاث حالات تفوق 41% من العينة التحكمية بالمقارنة بالجداول القياسية في العينة التكمية.

بينما بالنسبة للطول لوحظ أن متوسط الطول في المجموعات المرفعة 132,24 ± 17,89 كم في المجموعة التكمية ولم يمل هذا الاختلاف إلى دلالة إحصائية.

ولقد تراوح طول المجموعات المرفعة من 79,55 إلى 167 سم بينما تراوح بين 96,5 إلى 166 سم في المجموعة التكمية.

ولقد لوحظ أن الطول أقل من المعدل الخامس في 33 مريض (52,5%) بينما كان في الحدود الطبيعية بالمقارنة بالجداول القياسية ماعدا 32 حالة (41%) كان فوق المعدل الخامس والتسعين و12 حالة أخرى (14%) أقل من المعدل الخامس.

ولقد لوحظ أن النمو في الاناث قد تأثر أكثر من النمو في البالغين بالمرض خاصة الوزن والطول حيث كان له دلالة إحصائية في الإناث وليس في الذكور.

العمر في الذكور حينما تم تقسيم المجموعة المرفعة حسب طريقة بناء المريض وجدنا ان متوسط العمر الزمني 8,41 - 14,6 سنة لكل من حالات مرض.
البداية الجهازية ومرضى البداية قليلة المفاعل ومرضى البداية متعددة المفاعل على الترتيب، أيضاً متوسط الوزن كان 31,132,76 خ 17،17 وزن كجم ومتوسط الطول كان 116,9131,813 + 143,05 سم في المجموعات السابقة على الترتيب وكان لهذا الاختلاف دلالة إحصائية عالية.

وقد كان كل من الوزن والطول أقل من المعدل الخامس في 7 مرئي، تمثل 37% لكل منهما من مرضى البداية الجهازية (15 مرئي).

وقد كان كل من الوزن والطول أقل من المعدل الخامس في 4 مرئي تمثل 12% لكل منهما من مرضى البداية قليلة المفاعل (8 مرئي).

وقد كان الوزن أقل من المعدل الخامس في 13 مرئي تمثل 70% وكان الطول أقل من المعدل الخامس في 11 مرئي تمثل 64% من مرضى البداية متعددة المفاعل (17 مرئي).

وبالنظر لفترة المريض لاحظنا أن:

طول الباذ وبصمة الطرف العلوي والطرف السفلي قد تأثرت في المريض الذين عانو من المرضى لمدة تزيد على 5 سنوات ولكن هذا الاختلاف لم يكن له دلالة إحصائية.

وقد كان الوزن أقل من المعدل الخامس في 8 حالات تمثل 53% وكان الطول أقل من المعدل الخامس في 7 حالات تمثل 47% من المرضى الذين تقل مدة المرض فيهم عن 2 سنوات (15 مرئي).

بينما كان كل من الوزن والطول أقل من المعدل الخامس في 16 حالة تمثل 8 لكل منهما من المرضى الذين تزيد مدة المرض فيهم عن ثلاث سنوات (20 مرئي).

وإذن، تم مقارنة المرضى الذين تم علاجهم بمشتقات الكورتيزون (16 مرئي) والمريض الذين تم علاجهم بالعقاقير المشابهة للالتهابات الغير
محلوية على مشتقات الكورتيزون (24) مريغ وجد أن جميع المفاسات الإحصائية بفي المجموعة الأولى أقل من المجموعة الثانية ولكن هذا الاختلاف لم يكن له أي دلالة إحصائية.

- وقد وجد أن الوزن كان أقل من المعدل الخاص في 16 حالة (7،7%)
- بينما كان الطول أقل من المعدل الخاص في 14 حالة (8،8%)
- من المرضى الذين تم علاجهم بالعقار الفيضا للالتهاب الغير محتوية على مسقط الكورتيزون أو مثبتات الكورتيزون لمدة أقل من 1 أو تساوي 4 شهور (46 مريغ).

- وقد وجد أن كل من الوزن والطول كانا أقل من المعدل الخاص في 6 حالات (3،34%)
- لكسر من المرضى الذين تم علاجهم بمثبتات الكورتيزون أكثر من 4 شهور.

وفي النهاية امكن استنتاج أن مرضى الروماتويد الحديث قد تأخر في النمو في كل ابعاد النمو وان بعض هذه الابعاد كان له دلالة إحصائيّة (مثل الوزن وطول الاباع وعرض القدم اليمنى) خاصة في الاناث.

وايضاً وجد أن الاختلاف في النمو يزداد مع ازدياد مدة المرض وأيضاً يزداد هذا الاختلاف بإعطاء مركبات الكورتيزون.
دراسة قطعية انترروبومترية

مرض الروماتوид المفصلي الحدث

بحث مقدم من الطبيبة / ايمان محمد منير محمد كامل
بكالوريوس الطب والجراحة

 получить للحصول على درجة الماجستير

في الروماتيزم والتأهيل

تحت إشراف

١٠٠٠ عبد السعيد إبراهيم الحوال

استاذ ورئيس قسم الروماتيزم والتآهيل

كلية الطب - جامعة الزقاق

د. سامية محمد عبد المنعم

مدير الروماتيزم والتآهيل

كلية طب بنها

كلية طب بنها

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