DERMATOGLYPHIC PATTERNS

AS PREDICTORS OF TREATMENT RESPONSE IN

SCHIZOPHRENIA

Dissertation submitted in partial fulfillment of the

Degree of Master of Surgery (Anatomy)

Of

The Tamil Nadu Dr. M.G.R. Medical University,

Chennai

by

Dr. Aastha

CERTIFICATION

This is to certify that the dissertation titled **"**Dermatoglyphic patterns as predictors of treatment response in schizophrenia" *is based on the results of the work carried out by*

Dr. AASTHA

for the degree of Master of surgery (Anatomy) under my supervision. The work reported in this dissertation has not been submitted to any other university for the award of a degree.

Dr. Bina Isaac,
Professor of Anatomy,
Department of Anatomy,
Christian Medical College,
Vellore – 632 002.

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Dr. Sunil J Holla,
Professor and Head,
Department of Anatomy,
Christian Medical College,
Vellore – 632 002.

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CONTENTS

TITLE OF THE ABSTRACT	**:**	**Dermatoglyphic patterns as predictors of treatment response in Schizophrenia**
DEPARTMENT	**:**	**Anatomy**
NAME OF THE CANDIDATE	**:**	**Dr. Aastha**
DEGREE AND SUBJECT	**:**	**M.S. Anatomy**
NAME OF THE GUIDE	**:**	**Dr. Bina Isaac**

OBJECTIVES:

To identify,

1. Whether abnormal neurodevelopmental markers manifest more in treatment resistant schizophrenia, than in treatment responders by studying dermatoglyphic markers.
2. Whether treatment resistance can be predicted by examining dermatoglyphic variables.

METHODS:

Data are expressed as number (%) and mean +/- standard deviation for categorical and continuous variables.

Chi-square/Fisher's exact tests were used for group comparisons.

Independent t- test (for normal data) and Mann-Whitney test (for non-normal data) were performed to compare the mean scores between treatment responders and non- responders.

Pearson correlation analysis were done to assess the relationship between treatment responders and non- responders with respect to the ridge counts.

All the analysis were carried out using SPSS software Vs. 11.0.

RESULTS:

There was increased pattern frequency in the I_3 area of the left hand of non-responders and in the I_4 area of the right hand of responders. atd angle was decreased in the left hand of non-responders. There was increased frequency of the Simian type I crease in male non-responders and Sydney line in female non-responders. The t' position of axial triradius is the most common position in male non-responders. There is no significant correlation in the ridge counts of second and fourth digits of female non-responders and in a-b ridge counts in non-responders.

INTRODUCTION

Dermatoglyphics (from ancient Greek derma = "skin", glyphe = "carve") is the scientific study of fingerprints. The term was coined by Cummins and Midlo in 1926.

In humans, dermatoglyphics is present on fingers, palms, toes, and soles, and give insight into a critical period of embryogenesis, between 4 weeks and 5 months, when the architecture of the major organ systems is developing.

Dermatoglyphics has been studied for fortune telling by palmists and as a definitive and unalterable tool for identification by forensic experts. Widespread medical interest in epidermal ridges developed only in the last few decades when it became apparent that many patients with chromosomal aberrations had unusual ridge patterns. Inspection of skin ridges therefore promised to provide a simple, inexpensive means of information to determine whether a given patient could have a particular chromosomal defect (Schaumann and Alter, 1976).

Dermatoglyphics of many conditions like schizophrenia, Down's syndrome, diabetes mellitus, hypertension and epilepsy have been studied. Dr. Theodore J. Berry in his book "The hand as a mirror of systemic disease" has associated dermatoglyphics with 50 diseases or more, both congenital and acquired. Since most of the investigations needed to confirm the diagnosis in hereditary disorders are complex and expensive, dermatoglyphics can be efficiently employed with other clinical signs as a screening procedure to define indications for these laboratory procedures.

Dermatoglyphics offers atleast two major advantages as aid to the diagnosis of medical disorders.

i) The epidermal ridge patterns on the hand and soles are fully developed at birth and thereafter, remain unchanged for life.

ii) Scanning of the ridge patterns or recording these permanent impressions can be accomplished rapidly, inexpensively and without any trauma to the patient.

Schizophrenia is probably the most distressing and disabling mental disorder. The first signs of schizophrenia tend to surface in adolescence or young adulthood (Castle et al., 1991). People with schizophrenia suffer from problems with their thought processes that lead to hallucinations, delusions, disordered thinking, and unusual speech or behavior. It is a complex illness and scientists believe it is caused by a number of different factors that act together. These factors include: genetic influences, trauma (injury) to the brain occurring during or around the time of birth, as well as the effects of social isolation and/or stress. Schizophrenia affects between 1 and 2% of people (Hafner and Heiden, 1997). Whereas in men the illness tends to surface between the ages of 16 and 25, most females develop symptoms between the ages of 25 and 30.

Scientists believe the symptoms of schizophrenia are caused by abnormalities in the transfer and processing of information in the brain. Nerve cells in the brain communicate with each other by releasing chemicals called neurotransmitters from their nerve endings. Many of the symptoms of schizophrenia have been linked to abnormal activity of particular neurotransmitters. The nerve cells in question use two different neurotransmitters

called dopamine and serotonin, which play an important part in schizophrenia. The treatment of schizophrenia relies on drugs, the most important are the neuroleptics or antipsychotics. They modify the effects of the neurotransmitters in the brain. Interest has also focused on the neurotransmitter glutamate and the reduced function of the NMDA glutamate receptor in schizophrenia. It appears likely that multiple genes (Harrison et al., 2003) are involved in creating a predisposition to develop the disorder. In addition, factors such as prenatal difficulties like intrauterine starvation or viral infections (Brown, 2006), perinatal complications, and various nonspecific stressors, seem to influence the development of schizophrenia. Many studies of people with schizophrenia have found abnormalities in brain structure like enlargement of the ventricles (Johnstone et al., 1976), and decreased size or function of certain brain regions. Developmental neurobiologists have found that schizophrenia may result when neurons form inappropriate connections during fetal development. These errors may lie dormant until puberty, when changes in the brain that occur normally during this critical stage of maturation interact adversely with the faulty connections. Therefore it is believed that schizophrenia may be, in part, a disorder of the development of the brain.

Ectodermal ridges appear on the fingers and palm during the first and second trimester of pregnancy (Jim van OS et al., 2000). The rationale for studying dermatoglyphic features is derived from the fact that during their ontogeny, massive neural cell migration occurs in the brain, which is another ectodermal derivative. Thus, dermatoglyphic alterations in schizophrenia are

markers of disrupted early development and contribute support to the neurodevelopmental model of schizophrenia (Murray and Lewis, 1987; Bramon and Murray, 2001). They show that an insult, whether genetic, environmental or both, occurred during early mid-gestation (Green et al., 1994; Rosa et al., 2002).

The relevance of dermatoglyphics is not to diagnose, but to prevent by predicting a disease; not for defining an existing disease, but to identify people with the genetic predisposition to develop certain diseases.

AIMS OF THE STUDY

To identify,

1. Whether abnormal neurodevelopmental markers manifest more in patients with treatment resistant schizophrenia, than in treatment responders by studying the dermatoglyphic markers measured from finger and palm prints.
2. Whether treatment resistance can be predicted by examining the dermatoglyphic variables of an individual.

REVIEW OF LITERATURE

History of dermatoglyphics

Early History:

Cave drawings and petroglyph diagrams dating back thousands of years provide a record of early man's interest in hands, however the significance of these pre-historic samples is subject to broad interpretation.

Even the carving of Buddha (the statues in the museum of Calcutta) shows the presence of ridges on the palms and soles. But the scientific way of studying dermatoglyphics appears to be only since 1684. The first official mentioning of fingerprints was about the interesting markings found on the human fingertips in 1684. According, to Gray's Anatomy, absence of epidermal ridges occurs in less than 1% of people.

Some notable milestones in the history of dermatoglyphics are:

Date	Person	Historical Events
1685	Gouard Bidloo	First book with detailed drawings of fingerprints
1686	Marcello Malpighi	First observations of fingerprints under microscope
1788	J.C.A. Mayer	First to write out basic tenets of fingerprint analysis

1823	John E. Purkinje	First classification system of dermatoglyphics, nine print categories
1880	Dr. Henry Faulds	Suggests picking up fingerprints at crime scene
1883	Mark Twain	Dramatic fingerprints identification was introduced
1892	Sir Francis Galton	First practical method of fingerprint identification, responsible for basic nomenclature (arch, loop, whorl). Scientifically demonstrated permanence of fingerprints.
1897	Harris Hawthorne Wilder	First American to study dermatoglyphics; Named the A, B, C, D triradii points; invented the Main Line Index; studied thenar, hypothenar eminences, zones II, III, IV.
1904	Inez Whipple	First serious study of non-human prints.
1923	Kristine Bonnevie	First extensive genetic studies.

Embryogenesis of human epidermal ridges

Dermal ridge differentiation takes place early in fetal development. The resulting ridge configurations are genetically determined and influenced by environmental forces. It has been noted that the ultimate epidermal ridge patterns form at the sites of foetal volar pads. Fetal volar pads are mound-

shaped elevations of mesenchymal tissue, situated above the proximal end of the most distal phalanx on each finger. They are also located in each interdigital area, in the thenar and hypothenar areas of the palms and soles, and in the calcar area of the sole. The formation of these pads is first visible on the fingertips during the sixth to seventh week of development. The pads become very prominent during the subsequent several weeks, diminish again in the fifth month and disappear completely in the sixth month. Within this period, the dermal ridges coalesce into specific patterns, replacing the volar pads. The presence of volar pads as well as their size and position are responsible for the configuration of papillary ridge patterns, as postulated by Bonnevie (1924). For example, small pads would result in a simple pattern (arch), where as more prominent pads would tend to lead to the development of large and more complex systems of ridge configurations (loops and whorls). Similarly, foetal pads positioned symmetrically on the volar aspect of the fingertip would give rise to a pattern centered in the middle of the pattern area (whorl). Asymmetrical pads positioning leads to a pattern asymmetrically oriented within the pattern area (loop, either ulnar or radial according to the position of the pad). It has been established that the critical period of ridge formation begins in the foetus at approximately 70 mm crown rump length that is about, 3 months of age, when the volar pads are near or just beyond their peak development. The epidermal ridge patterns are completed only after the sixth prenatal month, when the glandular folds are fully formed and after the sweat gland secretion and keratinisation have begun. At this time the configurations on the skin surface

begin to reflect the underlying patterns. The surface epidermal furrows correspond to the furrow folds of stratum germinativum and each epidermal ridge is formed above a glandular fold. Ridge differentiation progresses from the apical pads proximally and in a radioulnar or tibiofibular direction (Loesch, 1986).

Several hypotheses have been formulated, concerning the forces that are responsible for the development of specific ridge patterns. Cummins speculated that the dermal ridge configurations were the result of physical and topographic growth forces. It is believed that the tensions and pressures in the skin during early embryogenesis determine the direction of the epidermal ridges (Schaumann and Alter, 1976). Bonnevie (1924) postulated that underlying arrangement of peripheral nerves may determine the direction of epidermal ridges. Penrose (1973) suggested that the ridges followed lines of greatest convexity in the embryonic epidermis. Hirsch and Schweichel (1973) have summarized present knowledge concerning the induction of the glandular folds and in turn the formation of epidermal ridges. Based on previous observations and their own studies, they pointed out to the regularity in the arrangement of the blood vessel nerve pairs under the smooth epidermis corium border. This situation exists shortly before formation of the glandular folds; they postulated that, the folds are induced by the vessel-nerve pairs. They list inadequate supply of oxygen to the tissues, deviations in the proliferation in the epithelial basal layer, and disturbances in keratinization of the epithelium as other factors that may influence epidermal ridge patterns. Even environmental factors such as

external pressure on the foetal pads and perhaps embryonic movements, particularly finger movement, can influence ridge formation.

The findings Mulvihill and Smith (1969) can be summed up as follows:

6 -8 weeks after conception	Volar pads form (these are little ball like structures, eleven per hand, that make up the contour of the developing fetal hand)
10 -12 weeks	Volar pads begin to recede
13th week after conception	Skin ridges (fingerprints) begin to appear, taking the shape of the receding volar pad
21st week after conception	Fingerprint patterns are complete

Methods of recording dermatoglyphics

A number of methods for recording dermatoglyphics exist. The methods vary in their requirements for equipments, time and experience and in the quality of prints produced.

Dermatoglyphic patterns are usually recognizable by the naked eye. A simple magnifying lens, preferably with a light source, helps greatly in scanning dermatoglyphics, especially in infants and small children, whose patterns are

very fine. Permanent impressions or prints are necessary for quantitative analysis of dermatoglyphics.

To enhance the quality of dermatoglyphic prints it is necessary to remove sweat, oil and dirt from the skin. This can be accomplished by washing the ridged areas with soap and water and with ethyl alcohol or ether.

Care must be taken to print the ridged areas completely. The ridges are primarily on the volar surface but also pass upwards and along the lateral margins of the fingers, palms, toes and soles. Therefore a print of only the volar surface may be incomplete and it is often necessary to roll the digits, palms and soles to ensure obtaining a print of the whole pattern. Palm prints must include, the area from the distal crease of the wrist to the metacarpophalangeal creases, and complete printing of both ulnar and radial sides of the ridged areas must be assured. Very fine ridges may be accentuated by a coloring agent, such as ink from felt pens (Schaumann and Alter, 1976).

Standard Methods

All the methods are relatively easy to use, rapid and inexpensive. However, they vary in the quality of the prints obtained. One of the following methods may be used.

Ink Method

This is the most widely used method. The necessary equipment consists of printer's ink, a roller, a glass or metal inking slab, a sponge rubber, and good quality paper preferably with a slightly glazed surface. It is not suitable for use with uncooperative children and those with very fine ridges. The prints obtained by this method are not always of sufficiently good quality to allow accurate counting of ridges (Schaumann and Alter, 1976).

Inkless Method

This method makes use of a commercially available patented solution and specially treated sensitized paper. It was described in detail by Walker (1957). It is not popular currently. The method is suitable for printing hands or feet with well-demarcated dermal patterns (Walker, 1957).

Transparent Adhesive Tape Method

In this method, the print is produced by applying a dry colouring pigment to the skin, and lifting it off with the transparent adhesive tape. The colouring agent may be coloured chalk, dust, India ink, standard ink, carbon paper, graphite stick or powdered graphite, common oil pastel crayon, etc. This method is inexpensive, rapid and easy to use with all types of patients. Prints are clear and not smudged. They can be preserved for an indefinite period of time (Schaumann and Alter, 1976).

Photographic Method

This technique is based on the principles of total internal reflection which occurs when an object is pressed against a prism. The magnified image is photographed by a Polaroid camera. It needs relatively expensive equipment. Recently, even ordinary photographic method has been tried out (Schaumann and Alter, 1976).

Special Methods

These methods are not widely used. However, they may have some advantages that the standard methods cannot offer, such as allowing the study of the correlation between the epidermal patterns and the underlying bone structures (radiodermatography), study of sweat pores (hygrophotography), or study of the spatial shape of the ridged skin areas, for example in primates (plastic mold method) (Schaumann and Alter, 1976).

Braganca and Pick have developed a method wherein, the region to be investigated is blackened with graphite smeared on a piece of cardboard. The print is taken by the tesa film and then adhered to a transparent film strip or photo printing foil. Such a negative could be enlarged five or six times.

An apparatus has been developed by Mull which can take finger and palm prints without any inking and can automatically count ridge numbers between two prints.

The History of Schizophrenia

The word "schizophrenia" is less than 100 years old. However the disease was first identified as a discrete mental illness by Dr. Emile Kraepelin in 1887 and the illness itself is generally believed to have accompanied mankind through its history. Written documents that identify schizophrenia can be traced to the old Pharaonic Egypt, as far back as the second millennium before Christ. Depression, dementia, as well as thought disturbances that are typical in schizophrenia are described in detail in the Book of Hearts. The Heart and the mind seem to have been synonymous in ancient Egypt. The physical illnesses were regarded as symptoms of the heart and the uterus and originating from the blood vessels or from purulence, fecal matter, a poison or demons.

Dr. Kraepelin used the term "dementia praecox" for individuals who had symptoms that we now associate with schizophrenia (Kraepelin, 1907). The Swiss psychiatrist, Eugen Bleuler, coined the term, "schizophrenia" in 1911. The word "schizophrenia" comes from the Greek roots schizo (split) and phrene (mind) to describe the fragmented thinking of people with the disorder (Liddell and Scott, 1980). Both Bleuler and Kraepelin subdivided schizophrenia into categories, based on prominent symptoms and prognoses. Over the years, those working in this field have continued to attempt to classify types of schizophrenia. Five types were delineated in the DSM-III: disorganized, catatonic, paranoid, residual, and undifferentiated. The first three categories were originally proposed by Kraepelin.

These classifications, while still employed in DSM-IV, have not shown to be helpful in predicting outcome of the disorder, and the types are not reliably diagnosed.

The evidence that schizophrenia is a biologically-based disease of the brain has accumulated rapidly during the past two decades. Recently this evidence has been also been supported with dynamic brain imaging systems that show very precisely the wave of tissue destruction that takes place in the brain that is suffering from schizophrenia.

A study done on “Genetic loadings in schizophrenia: a dermatoglyphic study.” by Balgir et al., (1993) showed that the dermatoglyphic features of isolated schizophrenics significantly differed from those of normals and also from those who have a positive family history of schizophrenia in the northwestern part of India, thus indicating the involvement of genetic factors in the etiology of schizophrenia.

Schizophrenia was earlier conceptualized as a chronic illness of early onset and progressive deterioration (dementia praecox), but promising treatments are available since the 1950s. In spite of the availability of clinically effective antipsychotics, treatment response in schizophrenia is partial or incomplete in about one-fifth to one- third treated cases (Conley, 1997). Factors contributing to this treatment resistance include patient factors (compliance, poor social support), illness factors (negative symptoms) and treatment factors (inadequate dose, inappropriate drugs) (Pantelis, 1996). The current clinical practice involves a trial of at least two antipsychotics from different chemical

classes for a period of 6-8 weeks and improvement rated on a standardized measure such as BPRS (Brief psychiatry rating scale) (Francis et al., 1996; Herz, 1997). If the improvement is inadequate as defined by the criteria for treatment resistance (Kane et al., 1988), then a trial of clozapine is warranted.

Kane's criteria for treatment resistant schizophrenia

1) Evidence of adequate previous medication trials (a minimum of three 6-week trials in the preceding 5 years with typical antipsychotics from at least two chemical classes at doses equivalent to at least 1000 mg/ day of chlorpromazine) without significant symptom relief.
2) Persistant positive psychotic symptoms, with rating scores of >4 on at least two of four positive symptom items (conceptual disorganization, suspiciousness, hallucinatory behaviour and unusual thought content) on the BPRS.
3) The presence of at least moderately severe illness, defined as a minimum total BPRS score of at least 4 on the clinical Global Impression- severity scale.
4) No period of good social or occupational functioning in the preceding 5 years.

The Kane's criteria were viewed as stringent and subsequent modifications were suggested, such as limiting duration of occupational dysfunction criteria to 2 years (Brenner et al., 1990), requirement of only two drug

trials to show resistance (Kinon et al., 1993), and so on. The Department of Psychiatry, CMC, takes into consideration the modified Kane's criteria and also individualistic decisions before considering a person treatment resistant and thereby starting clozapine. Clozapine may also be started for otherwise treatment responders but who may have developed serious adverse effects like tardive dyskinesia with other antipsychotics.

Neurodevelopmental factors associated with poor treatment response have been identified, like lower level of premorbid functioning, presence of deficit state, male gender, cavum septum pellucidum abnormalities (Murray et al., 1992), high prevalence of obstetrical complications (Weinberger, 1995), lateral and third ventricle enlargement (Lieberman et al., 1993), vulnerability to tardive dyskinesia (Chakos et al., 1996).

The variation of treatment resistance and treatment responding schizophrenics and their relationship with degree of neurodevelopmental disorders has not been studied. Also whether in future the dermatoglyphic variable can be used as one of the methods to predict treatment resistance in a case has not been established.

MATERIALS AND METHODS

Source of data

The material for the study consisted of finger and palm prints of outpatients and inpatients of the Mental Health Centre of the Christian Medical College, Vellore.

Study Groups

144 patients who were schizophrenics responding to treatment and 44 patients who were schizophrenics not responding to treatment were chosen for the study. The patients not responding to treatment were determined by the fact they were put on clozapine due to drug resistance or for other indications like tardive dyskinesia. The age group of the patients ranged from 18 to 60 years and both sexes were included.

Inclusion Criteria

All inpatients and outpatients attending the Psychiatry OPD on Tuesdays and Thursdays – diagnosed to have schizophrenia (F20. - category) by ICD- 10 criteria.

Exclusion Criteria

Patients with the following conditions were not included in the study:

1) Obstetrical complications

2) Untreated psychosis
3) Vulnerability to tardive dyskinesia due to other antipsychotics.

Patients were informed about the procedure in detail and their consent was obtained for the sudy.

The materials used were

1) Kores finger print ink
2) Ink Roller
3) Slab
4) Paper for taking the prints
5) Soap and water for removing the ink from the hands.

Method for recording finger and palm prints

Finger and palm prints of both hands were obtained by the following standardized method. Finger print ink was used for taking the dermatoglyphic prints. A drop or two of the semi-solid ink was poured on a clean slab.This ink was evenly spread on the glass plate by a roller. The patient touched the ink on the slab by placing the palm on its side and rolling it smoothly across the ink 180 degrees to the opposite side and lifting it. He/she repeated the motion on paper, transferring the print. The paper was placed at the edge of the table to give the patient’s hand space to rotate. The ink was removed from the hands with soap and water.

After the finger and palm prints were obtained by the above method, they were analysed qualitatively and quantitatively.The qualitative analysis done include, finger print patterns, patterns in palmar interdigital areas and palmar flexion creases. The quantitative analysis include, total and absolute finger ridge counts, a-b ridge count and atd angle.

Finger print patterns

Patterns on the finger-tips were classified by Galton, (1892) into three main types, depending on the number of triradii present. A useful descriptive term in dermatoglyphics is the triradius. A triradius (Fig. 1) is a point of convergence for three regions that separate almost parallel ridges.

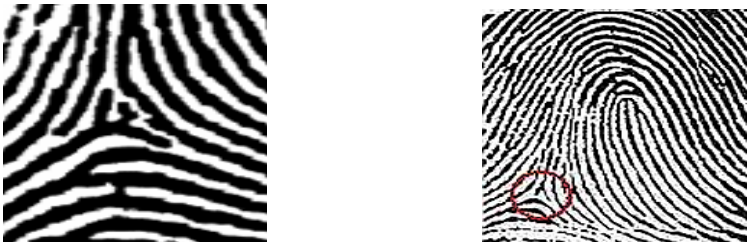

Fig.1 TRIRADII

The simplest pattern to be found on the fingertips is an arch. It has no triradius. (Fig. 2). The most common pattern on the fingertip is a loop. It has one triradius. It is of two types. If the ridge opens on the ulnar side, the resulting loop is termed an ulnar loop (L^u) (Fig. 3) whereas if it opens towards the radial margin, it is called a radial loop (L^r) (Fig. 4).

A whorl (W) in Galton's classification is any ridge configuration with two or more triradii. There are different types of whorls – simple (Fig. 5) and composite whorls (Fig. 6).

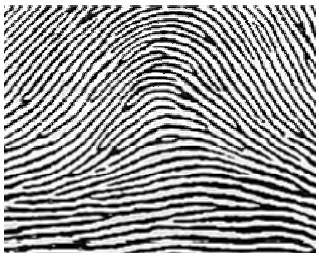

Fig. 2 PLAIN ARCH

Fig. 3 ULNAR LOOP

Fig. 4 RADIAL LOOP

Fig. 5 TRUE WHORL

Fig. 6 COMPOSITE WHORL

Total ridge count

A ridge count is made by drawing a line from the triradius to the center of the pattern and determining the number of intersected ridges between these two points (Fig. 7). Arches are defined as having a ridge count of zero. The ridge count of a whorl consists of the higher of the two counts. A total ridge count (TFRC) is the summation of the ridge counts for all the ten fingers. The triradius is not included in the count, nor is the final ridge when it forms the center of the pattern. Ridges which run close to the line without meeting it are excluded, but two ridges resulting from a bifurcation are both counted. Islands are counted. The total finger ridge count and the absolute finger ridge count (AFRC) are the same if no whorls are present. The TFRC expresses the size of pattern and the AFRC reflects the pattern size as well as its intensity (Weninger et al., 1976).

Fig. 7 TECHNIQUE OF RIDGE COUNTING. THIS LOOP HAS 13 RIDGES

At the base of the palm, there are usually four triradii: a, b, c and d. An axial triradius (t) is usually located near the point where the palm is connected to the wrist. Two percent of normal individuals have this triradius positioned near the center of the palm (termed t") (Fig. 8). A triradius found halfway in between these two positions (t') is found in 21% of the normal population.

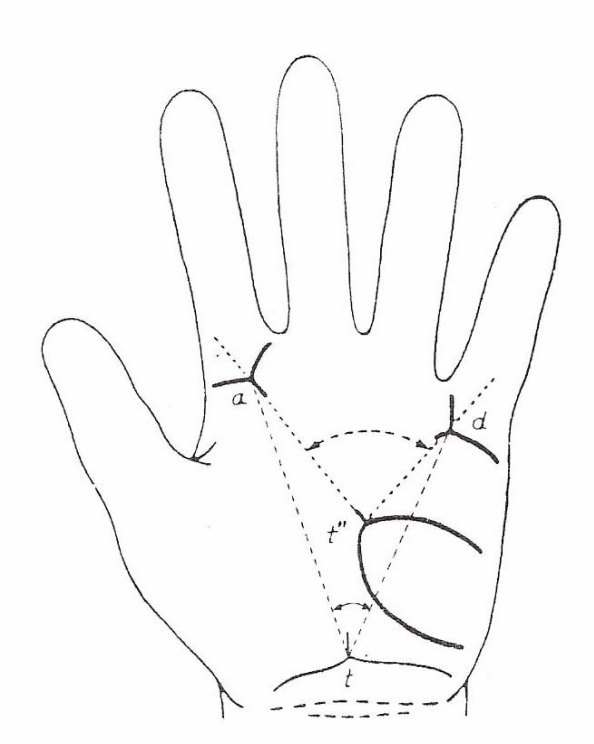

Fig. 8 AXIAL TRIRADII t, t', t'' & atd ANGLE MEASUREMENT

The atd angle is got by drawing a line from a to t and from d to t (Fig. 8). The atd angle averages 48° in normal individuals. This angle was used to measure the relationship of the length of the hand to its width. It is influenced by skeletal growth, which can continue into adolescence (Schaumann and Alter, 1976).

a-b ridge count

The ridge count most frequently obtained is the a-b ridge count. Counting was carried out along a straight line connecting the triradii ‘a’ and ‘b’ (Fig. 9).The count excludes the ridges forming the triradii. If there is an accessory triradius a’, counting is still done from triradius ‘a’ because it is the more radial of the two.

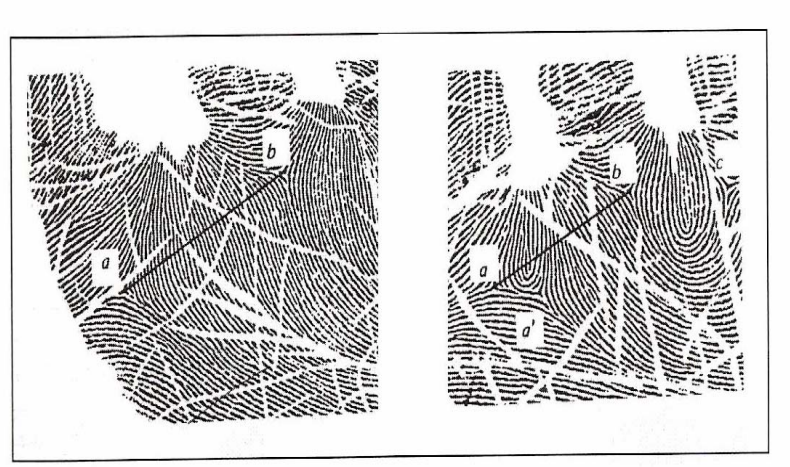

Fig. 9 a-b RIDGE COUNT

Palmar Pattern

Patterns on different areas of the palm (Fig. 10) are similar to those found on fingers, but usually larger and sometimes more complex. Thus, in the hypothenar area, the principal patterns are loops of various types, including S-shaped patterns made up of double loops, and whorls, often with three triradii.

Thenar patterns are frequently distinctive, incorporating loops, with some ridges running at right angles to the general ridge direction in the area. Inter-digital patterns in areas I_2, I_3, I_4 are almost invariably loops opening into the nearest inter-digital space. Rarely, very small whorls are found in this part of the palm.

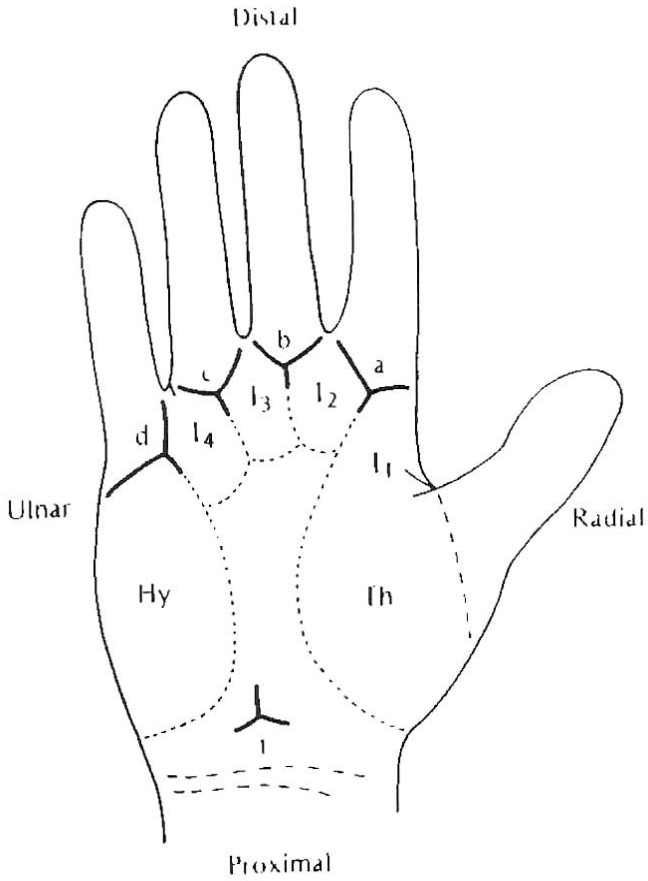

Fig. 10 DERMATOGLYPHIC PALMAR PATTERN AREAS

Palmar flexion creases

These creases represent the location of the firmer attachment of the skin to underlying structures. Palmar creases are of 2 types - major & minor. Here only major creases are considered. The first to appear is the radial longitudinal crease that borders the thenar eminence. This is followed by the proximal transverse crease (PTC), and distal transverse crease (Fig. 11).

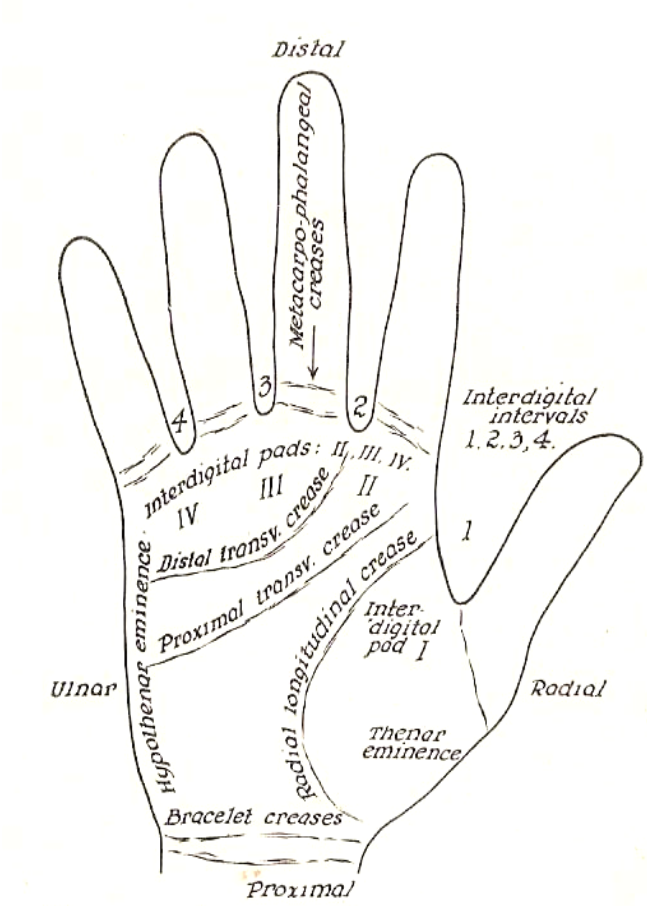

Fig. 11 NORMAL PALMAR FLEXION CREASES

Sometimes the proximal and distal transverse creases are replaced by or joined into one single crease that traverses the whole palm. This single transverse flexion crease is usually referred to as the Simian crease or line (Fig. 12). Variants of single palmar crease have been noted (Fig. 13). They are Simian transitional type I (proximal and distal creases connected by a bridging crease) and Simian transitional type II (fusion of the transverse creases with branching proximal and distal segments). A variation in appearance of PTC is the Sydney Line (SL) (Fig. 14) after the city in Australia where it was observed first. SL represents PTC extending beyond the hypothenar eminence to the ulnar margin of the palm. The distal transverse crease persists and that appears normal (Purvis Smith, 1969).

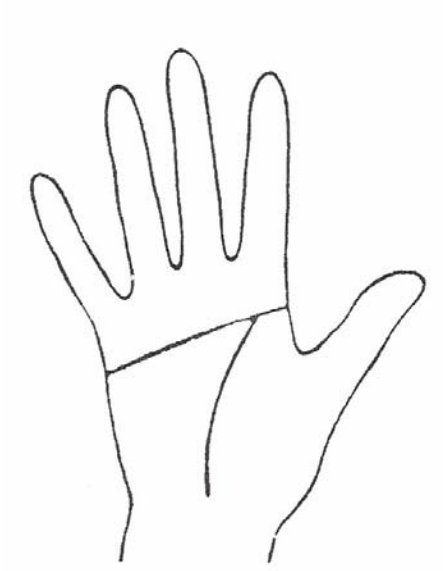

Fig. 12 SINGLE TRANSVERSE CREASE OR SIMIAN CREASE

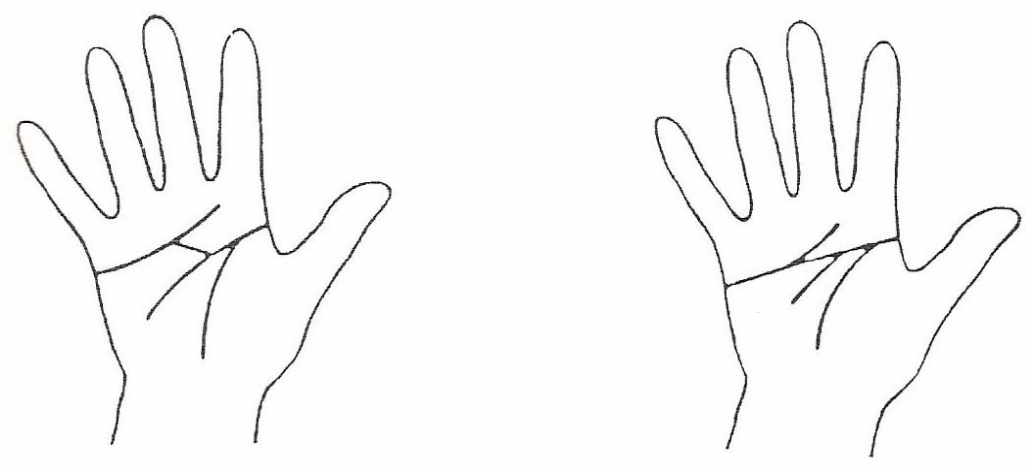

Fig. 13 SIMIAN TRANSITIONAL TYPE I & TYPE II

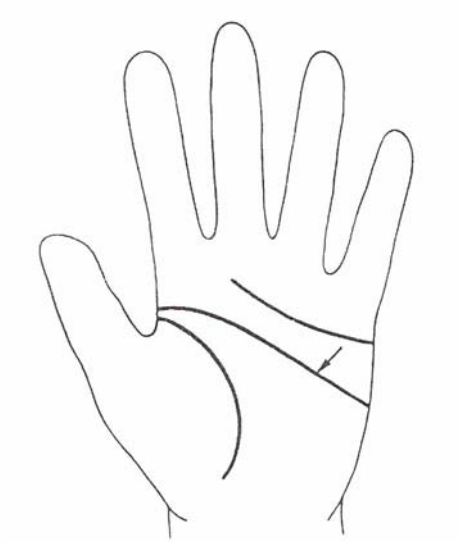

Fig. 14 SYDNEY LINE

Pattern symmetry and asymmetry

The occurence of like finger-print types in the individual referred to by Galton (1892) as “the tendencies of digits to resemble one another,” is termed *association* by Waite, (1915). Biologists now designate genetic association of any traits as *pleiotropy*.

Waite (1915) found in 2000 males, 12% have patterns of the same type-arches, loops, whorls on all ten digits, 16% of the persons in the Waite’s series have nine patterns of the same type, and 10% have eight patterns of the same type. The incidence of complete association in single hands is naturally higher than that in individuals, since there are five instead of ten digits concerned. Correlations suggest a generalized tendency in the apical volar pads of the fetus to behave in their development and regression as if they were under a common control. Positive correlations of pattern occurrences indicate that all areas are to some extent subject to a common control, rigid enough to determine not only the existence of patterns, but also to regulate the pattern type. The negative correlation must indicate the existence of superimposed local factors counteracting the effect of the more generalized control.

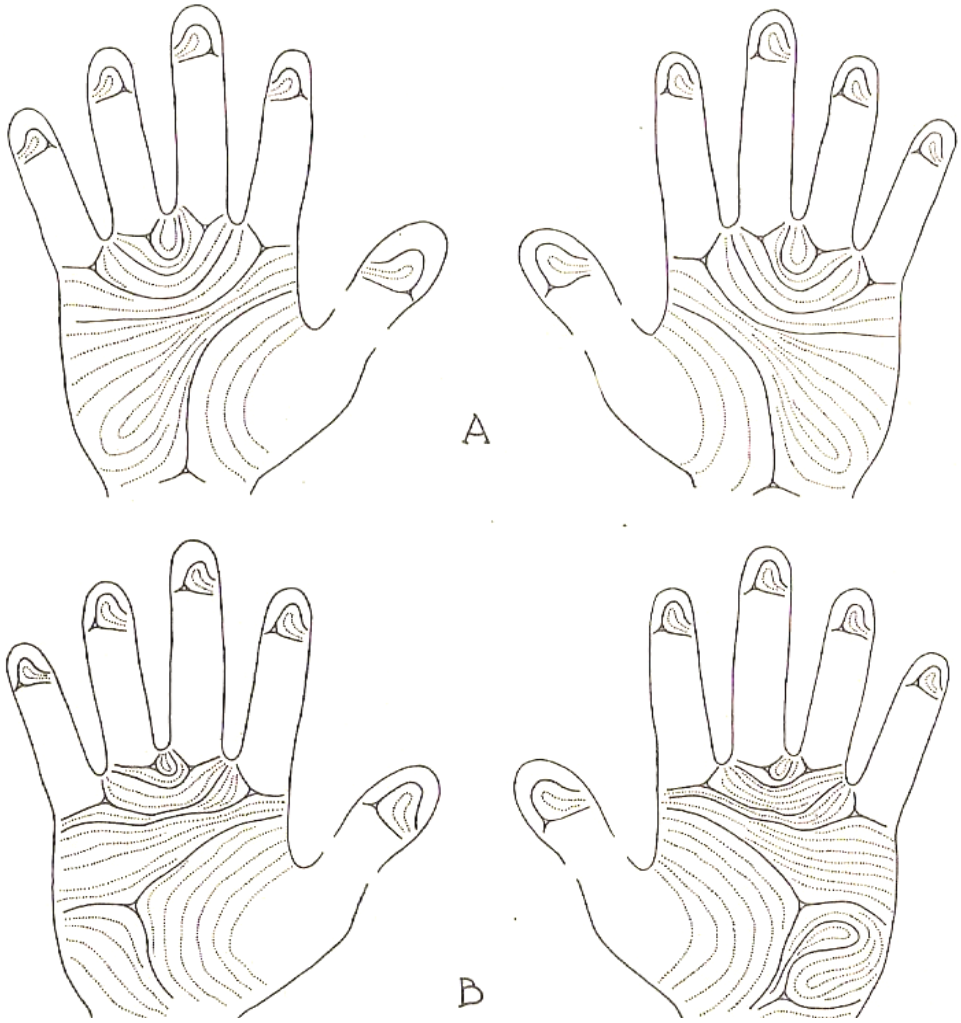

Fig. 15 A-BILATERAL SYMMETRY B-BILATERAL ASYMMETRY

Fig. 16 FULL HAND PRINT

Fig. 17 ARCH

Fig. 18 LOOP

Fig. 19 SIMPLE WHORL

Fig. 20 COMPOSITE WHORL

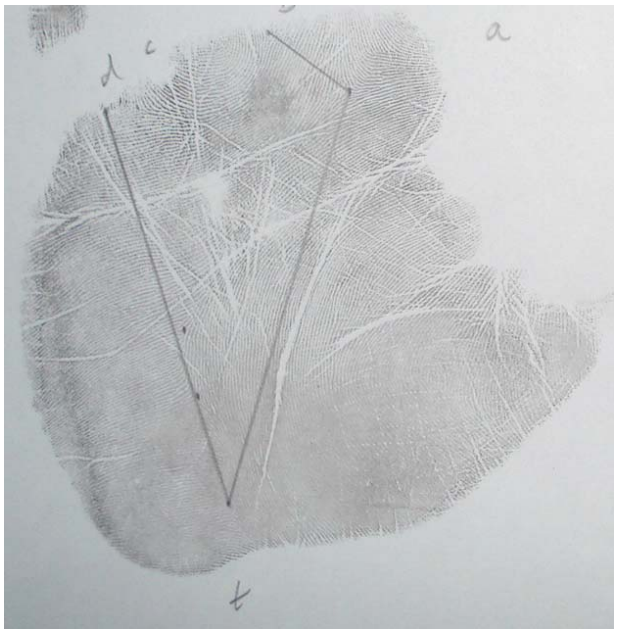

Fig. 21 AXIAL TRIRADIUS t

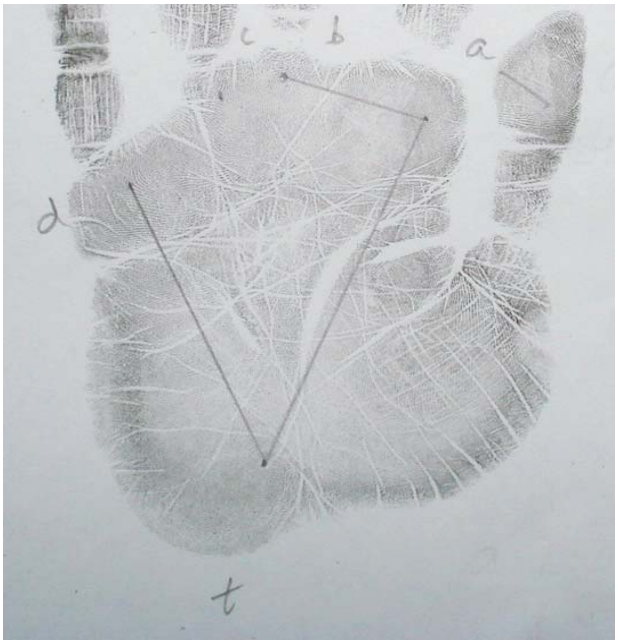

Fig. 22 atd ANGLE & a-b RIDGE COUNT

Fig. 23 THENAR, I_2, I_3, I_4, HYPOTHENAR PALMAR PATTERNS

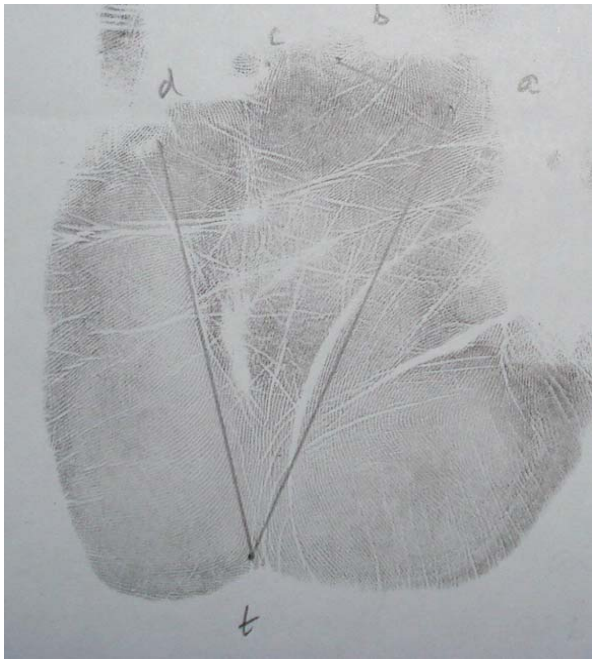

Fig. 24 NORMAL FLEXION CREASES

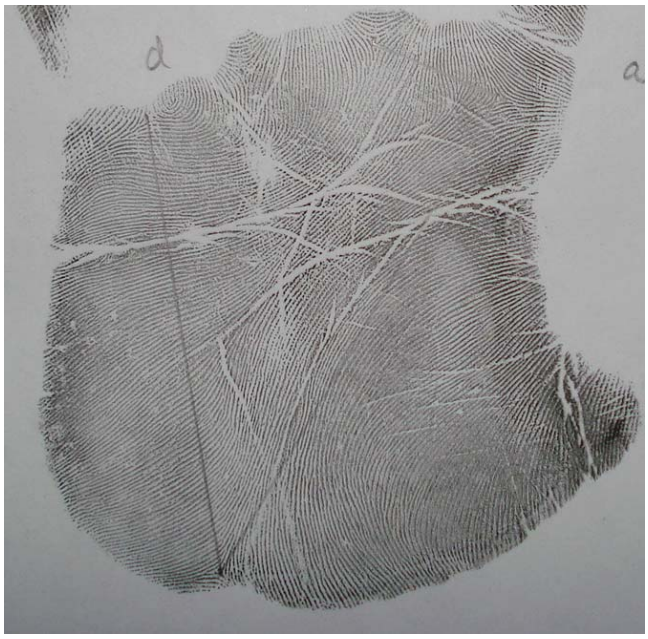

Fig. 25 SIMIAN TRANSITIONAL TYPE I

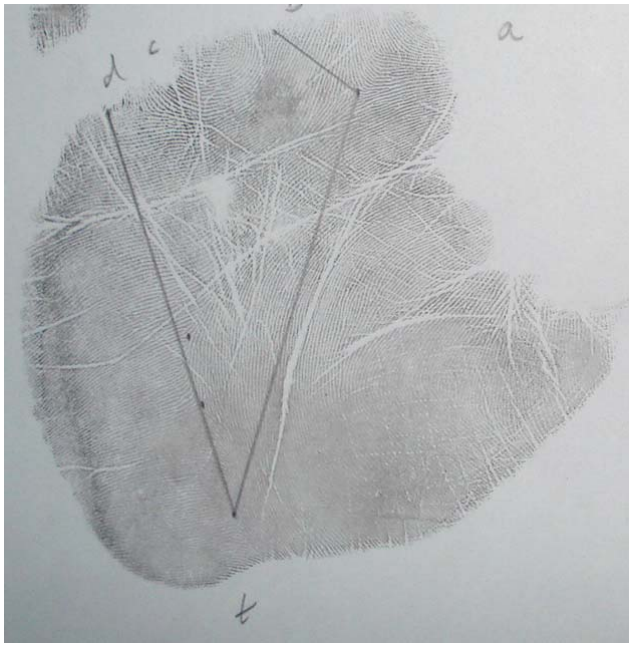

Fig. 26 SYDNEY LINE

Statistical Analysis

Data are expressed as number (%) and mean +/- standard deviation for categorical and continuous variables.

Chi-square/Fisher's exact tests were used for group comparisons.

Independent t-test (for normal data) and Mann-Whitney test (for non-normal data) were performed to compare the mean scores between treatment responders and non- responders.

Pearson correlation analysis were done to assess the relationship between treatment responders and non- responders with respect to the ridge counts.

All the analysis were carried out using SPSS software Vs. 11.0.

Table 1: Finger print pattern in treatment responders

Pattern	Treatment Responders (N = 144)	
	n	%
Arches	120	8.28
Loops		
Ulnar	805	55.68
Radial	27	1.84
Whorls		
True	397	27.26
Composite	101	6.91

The table shows frequency of percentage of finger print pattern in treatment responders, 8.28% have arches, 55.68% have ulnar loops, 1.84% have radial loops, 27.26% have true whorls and 6.91% have composite whorls.

Table 2: Finger print pattern in treatment non-responders

Pattern	Treatment Non-Responders (N = 44)	
	n	%
Arches	32	7.23
Loops		
Ulnar	248	56.10
Radial	8	1.80
Whorls		
True	122	27.60
Composite	32	7.23

The table shows frequency of percentage of finger print pattern in treatment non-responders, 7.23% have arches, 56.10% have ulnar loops, 1.80% have radial loops, 27.60% have true whorls and 7.23% have composite whorls.

Table 3: Frequency of finger print pattern in the two study groups

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Arches	120	8.28	32	7.23	0.823
Loops					
Ulnar	805	55.63	248	55.68	0.985
Radial	27	1.84	8	1.80	0.956
Whorls					
True	397	27.26	122	27.60	0.888
Composite	101	6.91	32	7.23	0.817

The table compares the frequency of finger print pattern in the 2 groups. The frequency of arches is slightly more in treatment responders than in non-responders but it was not statistically significant. The frequency of ulnar loops as compared to radial loops is more and it is statistically significant, (p=0.000) as shown in table 4. The frequency of composite whorls is slightly more in treatment non-responders but it was not statistically significant.

Table 4: Frequency of loops and whorls

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Loops					
Ulnar	805	55.63	248	55.68	0.000 *
Radial	27	1.84	8	1.80	
Whorls					
True	397	27.26	122	27.60	0.893
Composite	101	6.91	32	7.23	

* P value <0.05

Fig. 27 Frequency of distribution of finger Patterns.

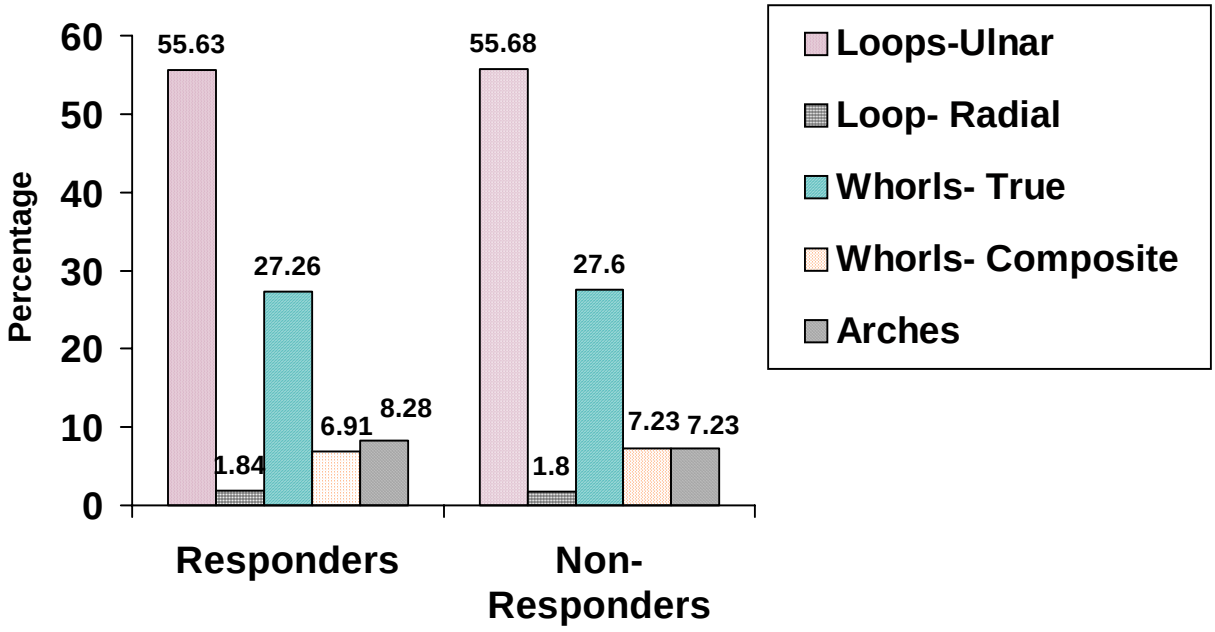

Table 5: Frequency of Hypothenar pattern in treatment responders and non-responders

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Right	44	30.6	13	29.5	0.890
Left	28	18.8	14	31.8	0.070

The frequency of hypothenar pattern in the right hand of the treatment responders was slightly more than the right hand of the non-responders but it was statistically not significant. The frequency of the hypothenar pattern in left hand of the treatment non-responders was found to be higher than the left hand of responders.

Fig. 28 Frequency of Hypothenar Pattern

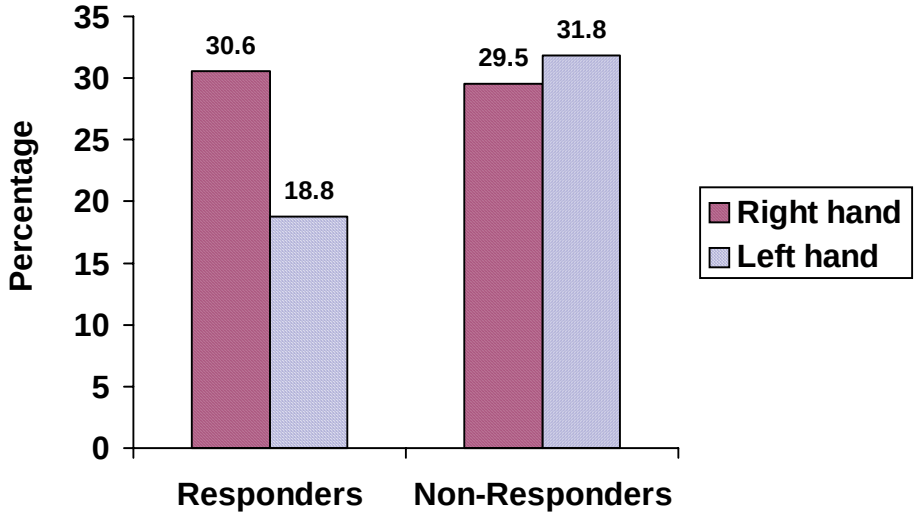

Table 6: Frequency of Thenar / I_1 pattern area in treatment responders and non-responders

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Right	26	18.1	6	13.6	0.490
Left	43	29.9	12	27.3	0.740

The frequency of thenar / I_1 in treatment responders was higher than that of non-responders, but it was not statistically significant.

Fig. 29 Frequency of Thenar/I_1Pattern area

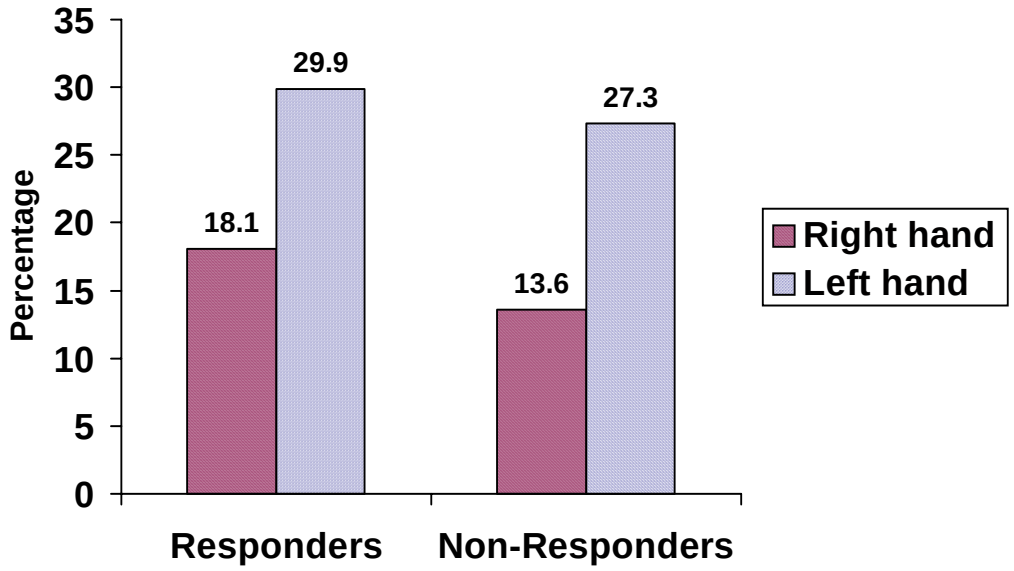

Table 7: Frequency of I_2 pattern area in treatment responders and non-responders

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Right	97	67.4	24	54.5	0.118
Left	125	86.8	35	79.5	0.234

The frequency of I_2 pattern in the treatment responders was higher than that of the non-responders, but it was not statistically significant.

Fig. 30 Frequency of I_2 Pattern Area

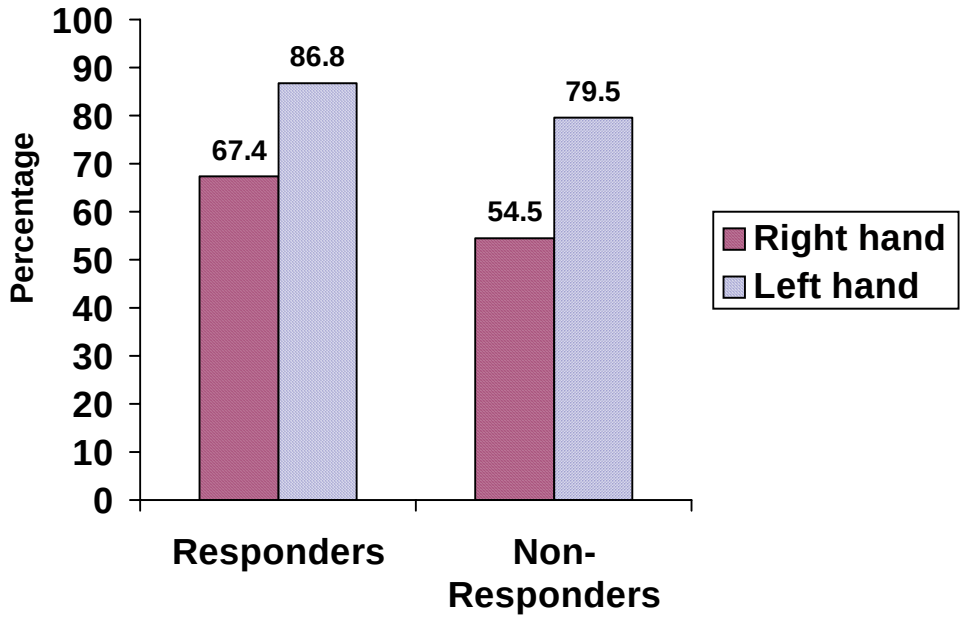

Table 8: Frequency of I_3 pattern Area in treatment responders and non-responders

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Right	84	58.3	23	52.3	0.482
Left	66	45.8	28	63.6	0.039 *

*P value <0.05

The frequency of I_3 pattern in the left hand of non-responders was higher than in the left hand of the treatment responders and it was statistically significant.

Fig. 31 Frequency of I_3 Pattern Area

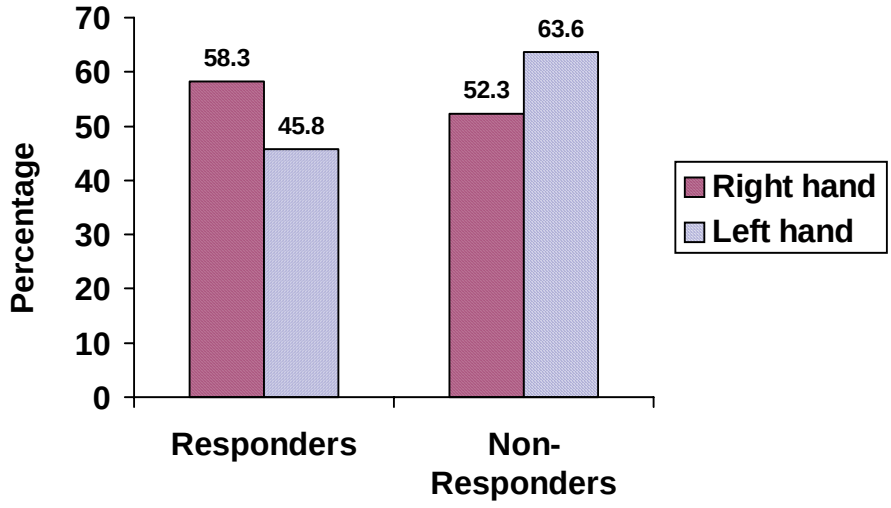

Table 9: Frequency of I_4 area pattern in treatment responders and non-responders

Pattern	Treatment Responders (N = 144)		Treatment Non-Responders (N = 44)		P value
	n	%	n	%	
Right	125	86.3	32	72.7	0.027 *
Left	104	72.2	34	77.3	0.503

*P value <0.05

The frequency of I_4 pattern in the right hand of the treatment responders was higher than the right hand of the non-responders, which was statistically significant.

The frequency of I_4 pattern in the left hand of treatment non-responders was higher than the left hand of the responders, which was statistically not significant

Fig. 32 Frequency of I_4 Pattern Area

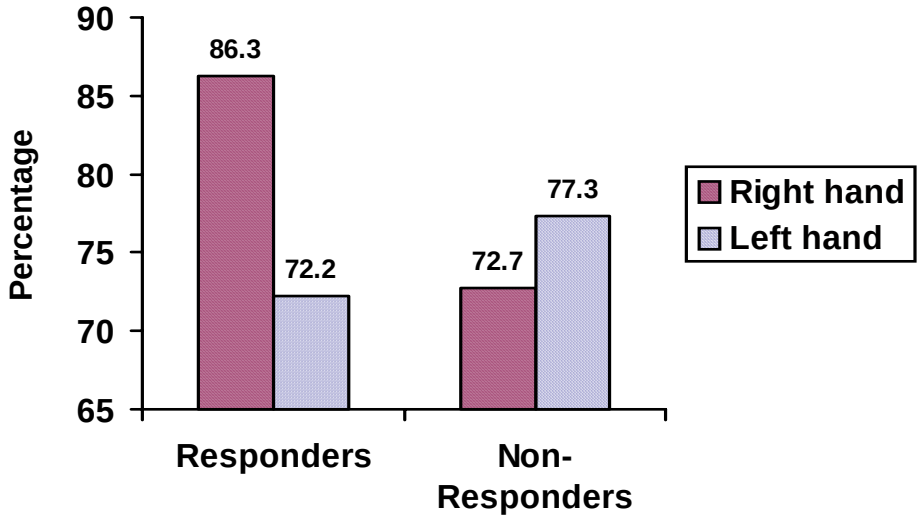

Table 10: Total finger ridge count in treatment responders and non-responders

	Number	Mean	SD	P value
Treatment Responders	144	184.4	59.7	0.470
Treatment Non-responders	44	177.1	53.2	

The mean of the total finger ridge count was higher in treatment responders than in non-responders, but was statistically not significant

Table 11: Absolute finger ridge count in treatment responders and non-responders

	Number	Mean	SD	P value
Treatment Responders	144	231.9	95.4	0.637
Treatment Non-responders	44	224.4	83.6	

The mean of the absolute finger ridge count was higher in treatment responders than in treatment non-responders, but was not statistically significant.

Table 12: atd angles in treatment responders and non-responders

	Treatment Responders (N = 144)	**Treatment Non-responders (N = 44)**
Right Hand		
Mean	38.96	38.56
SD	5.07	5.56
P Value	0.645	
Left Hand		
Mean	40.00	36.91
SD	6.79	3.83
P Value	0.004 *	

* P value <0.05

The mean atd angle in the right hand of the responders was nearly the same as that of the right hand of the non-responders and it was not statistically significant.

The mean atd angle in the left hand of the non-responders was less than that of the responders and it was statistically significant.

Table 13: Frequency of Palmar Flexion Creases in treatment responders and non-responders

	Male					Female				
	Treatment Responders (N = 86)		Treatment Non-responders		P-Value	Treatment Responders (N = 58)		Treatment Non-responders		P-Value
	n	%	n	%		n	%	n	%	
Left										
ST - I	16	18.6	17	48.6	0.0008*	9	15.5	3	33.3	0.194
ST - II	2	2.3	-	-	-	-	-	-	-	-
SL	7	8.1	5	14.3	0.3003	8	13.8	4	44.4	0.0259 *
SFC	3	3.5	-	-	-	-	-	-	-	-
Normal flexion creases	58	67.4	13	37.1	0.0022*	41	70.7	2	22.2	0.0047 *
Right										
ST - I	19	22.2	20	57.1	0.0002*	7	12.1	3	33.3	0.097
ST - II	1	1.2	-	-	-	-	-	-	-	-
SL	8	9.3	5	14.3	0.421	6	10.3	4	44.4	0.0075 *
SFC	1	1.2	1	2.9	0.511	-	-	-	-	-
Normal flexion creases	57	66.3	9	25.7	0.000*	45	77.6	2	22.2	0.0007 *

*P< 0.05

Frequency of ST-1 crease in both hands of the treatment non-responders (males) was higher than in the treatment responders and this was statistically significant.

Frequency of Sydney line was higher in both hands of treatment non-responders (females) than in the responders and this was statistically significant.

Fig. 33 Abnormal Palmar Creases Right hand - Male

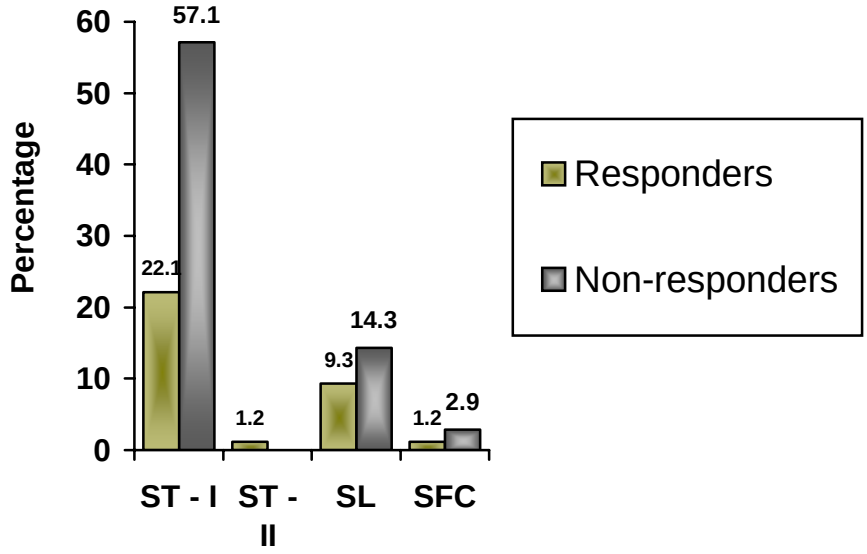

Fig. 34 Abnormal Palmar Creases Left hand - Male

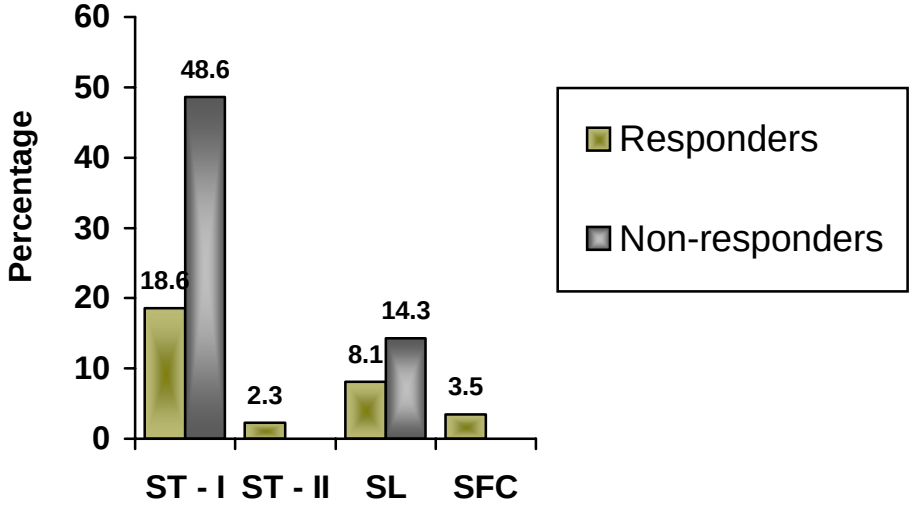

Fig. 35 Abnormal Palmar Creases Right hand - Female

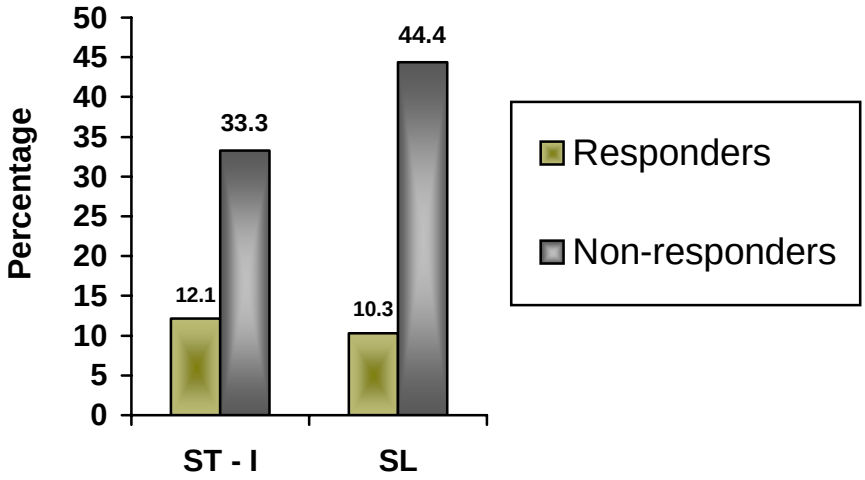

Fig. 36 Abnormal Palmar Creases Left hand - Female

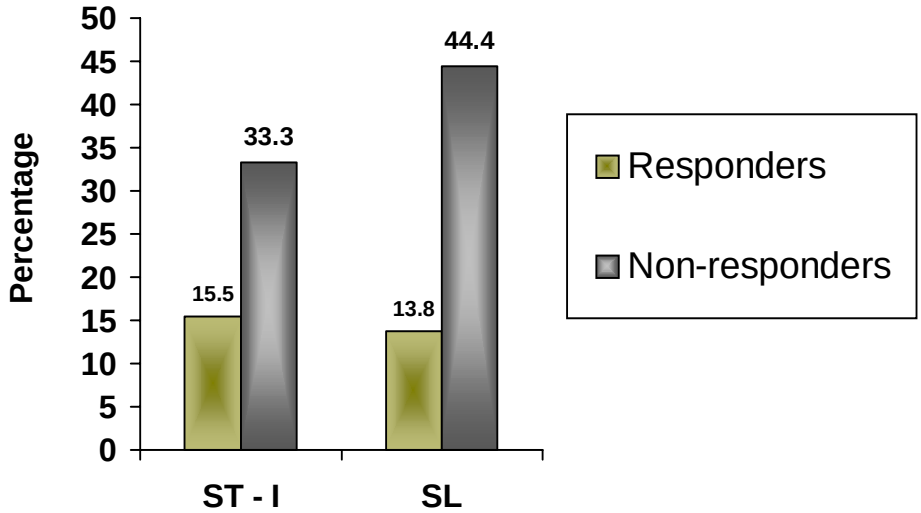

Table 14: Frequency of palmar flexion creases in the right and left hands of the treatment responders and non-responders

	Abnormal Flexion Creases					Normal Flexion Creases				
	Treatment Responders (N = 144)		Treatment Non-responders (N = 44)		P-Value	Treatment Responders (N = 144)		Treatment Non-responders (N = 44)		P-Value
	n	%	n	%		n	%	n	%	
Right hand	42	29.0	102	71.0	0.000*	33	75.0	11	25.0	0.000 *
Left hand	45	31.0	71	68.8	0.000*	29	66.0	15	34.0	0.000 *

*P value <0.05

The frequency of abnormal palmar flexion creases in the right and left hands in the treatment non-responders was higher than in the right and left hands in the responders and it was statistically significant.

Table 15: Frequency of abnormal palmar flexion creases in the right and left hands of the treatment responders and non-responders - Males

	Treatment Responders (N = 86)		Treatment Non-responders (N = 35)		P-Value
	n	%	n	%	
Right hand	19	22.1	20	57.1	0.000 *
Left hand	16	18.6	17	48.6	0.000 *

*P value < 0.05

The frequency of abnormal palmar flexion creases in both hands of the males among the treatment non-responders was higher than in both hands of the males among the responders and it was statistically significant.

Table 16: Frequency of palmer flexion creases in the right and left hands of the treatment responders and non-responders - Females

	Treatment Responders (N = 58)		Treatment non-responders (N = 9)		P-Value
	n	%	n	%	
Right hand	13	22.4	7	77.8	0.0007 *
Left hand	17	29.3	7	77.8	0.0047 *

* P value < 0.05

The frequency of abnormal palmar flexion creases in both hands of the females among the treatment non-responders was higher than in both hands of the females among the responders and it was statistically significant.

Table 17: Frequency of t-axial triradii position in treatment responders and non-responders

	Male					Female				
	Treatment Responders (N = 86)		Treatment Non-responders (N=35)		P-Value	Treatment Responders (N = 58)		Treatment Non-responders (N=9)		P-Value
	n	%	n	%		n	%	n	%	
Left										
t	80	93.0	29	82.9	0.092	49	84.5	8	88.9	0.7302
t’	2	2.3	5	14.3	0.0108 *	3	5.2	1	11.1	0.4878
t + t’	4	4.7	-	-	-	5	8.6	-	-	-
t + t’ +t”	-	-	1	2.9	-	1	1.7	-	-	-
Right										
t	79	91.9	30	85.7	0.3003	47	81.0	9	100.0	0.1525
t’	2	2.3	5	14.3	0.0108 *	2	3.4	-	-	-
t + t’	4	4.7	-	-	-	8	13.8	-	-	-
t + t’ +t”	1	1.2	-	-	-	1	1.7	-	-	-

*P value<0.05

t’ position was the most common position of the axial triradius in both hands of the male non-responders when compared to the responders and this was statistically significant.

Table 18: Correlation between the ridge counts of the digits of the right and left hands in treatment responders

	Male		Female	
	Correlation	**P value**	**Correlation**	**P value**
Ridge count				
D I	0.611	0.000 *	0.758	0.000 *
D II	0.367	0.000 *	0.648	0.000 *
D III	0.549	0.000 *	0.738	0.000 *
D IV	0.563	0.000 *	0.660	0.000 *
D V	0.638	0.000 *	0.724	0.000 *

* P value <0.05

The correlation between the ridge counts of the digits of the right and left hands in the treatment responders was statistically very significant.

Table 19: Correlation between the ridge counts of the digits of the right and left hands in treatment non-responders

	Male		Female	
	Correlation	**P value**	**Correlation**	**P value**
Ridge count				
D I	0.590	0.000 *	0.779	0.000 *
D II	0.796	0.000 *	0.527	0.145
D III	0.627	0.000 *	0.668	0.049 *
D IV	0.637	0.000 *	0.515	0.156
D V	0.550	0.000 *	0.672	0.048 *

*P value <0.05

The correlation between the ridge counts of the digits of the right and left hands in the treatment non-responders is statistically significant only in digits I, III and V.

Table 20: Correlation between the a – b ridge counts and atd angles of the right and left hands in treatment responders

	Male		Female	
	Correlation	**P value**	**Correlation**	**P value**
a –b Ridge count	0.664	0.000 *	0.428	0.001 *
atd angle	0.352	0.001 *	0.621	0.000 *

* P value <0.05

The correlation between the a-b ridge counts and atd angles of the right and left hands in the treatment responders was statistically significant.

Table 21: Correlation between the a – b ridge counts and atd angles of the right and left hands in treatment non-responders

	Male		Female	
	Correlation	**P value**	**Correlation**	**P value**
a – b Ridge count	- 0.013	0.939	0.307	0.421
atd angle	0.481	0.003 *	0.734	0.024 *

* P value <0.05

The correlation between the atd angles of the right and left hands in the treatment non-responders was statistically significant.

Table 22: Correlation between the various parameters of the right & left hands in treatment responders & non-responders - Males

	Responders (N = 86)		Non-responders (N= 35)		P value
	Mean	**SD**	**Mean**	**SD**	
atd angle					
Right	38.3	3.9	37.9	5.5	0.661
Left	39.3	7.0	36.4	3.9	0.022 *
a – b ridge count					
Right	46.9	11.1	43.9	10.3	0.179
Left	46.6	8.7	46.6	9.3	0.989
TFRC	184.3	59.0	177.3	52.9	0.543
AFRC	236.6	93.7	226.9	85.9	0.599

* P value <0.05

The correlation between the atd angle of the left hand in the treatment responders and non-responders only was statistically significant.

Table 23: Correlation between the various parameters of the right & left hands in treatment responders & non-responders – Females

	Responders (N = 58)		Non-responders (N= 9)		P value
	Mean	**SD**	**Mean**	**SD**	
atd angle					
Right	39.9	6.4	41.0	5.2	0.635
Left	41.0	6.4	38.9	2.8	0.331
a – b ridge count					
Right	46.6	9.9	49.4	5.3	0.213
Left	47.9	9.4	45.0	6.5	0.372
TFRC	184.6	61.2	176.6	57.8	0.715
AFRC	225.2	98.1	214.8	78.1	0.763

DISCUSSION

The study of dermatoglyphic abnormalities has been employed in a number of chromosomal disorders, congenital malformations, and neurological and neuropsychiatric conditions in order to gain insight into the complex interaction of environmental, developmental, and genetic factors leading to disease (Chakraborty 1991; Schaumann and Optiz 1991). Schizophrenia, diabetes mellitus, congenital heart disease, Down's syndrome (Rajangam et al., 1995), fragile X syndrome (Loesch et al., 2002), Brachmann-deLange syndrome (Barr et al., 1971), bipolar disorder (Torrey, 1999), leukaemia and thalassemia are a few conditions associated with dermatoglyphic abnormalities. The malformations of dermatoglyphic characteristics can result from a number of physiological insults that can occur during fetal development, including exposure to environmental toxins, viral infections, or genetic mutations.

Schizophrenia is a chronic recurring illness that is prominently characterized by reality distortions (hallucinations and delusions), thought disorder, cognitive dysfunction and negative symptoms. Skin and brain develop from the same ectoderm (van Oel et al., 2001) and cells migrate to the cortex at this time. It is probable that an insult causing damage to one of these systems would damage the other, and there is now evidence relating aspects of the dermatoglyphic profiles of the hands to schizophrenia (van Oel et al., 2001; Fearon et al., 2001). While there is compelling evidence of a genetic component (Gottesman and Shields, 1972) in the development of schizophrenia, the precise nature of this has yet to be fully elucidated. Much evidence also exists that

environmental factors acting during the prenatal and perinatal periods exert an effect that predisposes to the subsequent development of schizophrenia (Murray et al.1992). The second trimester of foetal development is particularly implicated as a period of potential vulnerability (Mednick et al., 1998). This trimester is the critical period for both foetal brain and epidermal ridge development. Thus, dermatoglyphics provide an attractive set of potential markers, as their development is temporarily localized to that period during which the maturing foetal brain may be at highest risk for the later development of schizophrenia.

Finger tip patterns (Arches, Loops, and Whorls)

Schizophrenics are found to exibit simpler fingertip patterns (Turek, 1990; Fananas et al., 1996; Avila et al., 2003). In the present study, there is no difference in the frequency of the finger tip patterns between the treatment responders and non-responders. The ulnar loops occurred more frequently than radial loops in both groups.

Hypothenar Area

The frequency of the hypothenar pattern was higher in the left hand of treatment non-responders than in treatment responders.

Thenar/I_1 Area

Usually, in most individuals there is no pattern in this area, but the ridges follow a mild course around the base of the thumb. Patterns when present are most often loops (Schaumann and Alter, 1976). The pattern frequency in the thenar/ I_1 area showed no significant difference between the two groups.

I_2 Area

The pattern frequency in the I_2 area showed no significant difference between the two groups.

I_3 Area

The pattern frequency in the I_3 area of the left hand of treatment non-responders was higher than in the responders.

I_4 Area

Fearon et al., (2001), in their study on 148 schizophrenic patients responsive to treatment, found decreased frequency of patterns in the right fourth interdigital area. In the present study, the pattern frequency in the I_4 area of the right hand of treatment responders was higher than in the non-responders.

Total and absolute finger ridge counts

Fananas et al., (1996) and Davis and Bracha (1996) found no significant difference in total finger ridge count in the sample they studied of schizophrenics who were responsive to treatment. Turek (1990) reported a decrease in TFRC in a large sample of responsive schizophrenic patients. In the present study, there is no difference in TFRC between the study groups. TFRC appears to be under relatively strong genetic control (Holt, 1968) and little influenced by environmental events.

a-b ridge count

Fananas et al., (1996) and Fearon et al., (2002) found a reduced total a-b ridge count a reliable marker of prenatal disturbance during the second trimester of life in their study of schizophrenic patients responsive to treatment. In the present study, there was no difference in the a-b ridge counts of treatment responders and non-responders. Rose et al., (1987) have suggested that the a-b ridge count is sensitive to environmental stress because the area of the palm in which the a–b ridge count is situated, the second interdigital region, begins to develop earlier than the fingers. However, ridge formation progresses more slowly on the palms than the fingers, and ridge differentiation proceeds in a distal radial to proximal ulnar direction. Thus, the ridges in the second interdigital region may develop over a longer period, exposing that area for a longer period to potential environmental insults. Davis and Bracha (1996) found evidence of asymmetrical development, with a-b ridge count reduction on the right whereas Fearon et al., (2001) found a marked reduction in the a-b ridge count on the left side, both studies being in patients responsive to treatment. In the present study, there was no such difference in the a-b ridge counts between the right and left hands in the two groups.

atd angle

Penrose (1954) observed that the atd angle is even more sensitive to environmental effects than the a–b ridge count. When the atd angles were compared between treatment responders and non-responders, statistically significant result was obtained for the left hand. The atd angles were decreased

in treatment non-responders i.e. around 36.9^{o} when compared to treatment responders i.e. around 40^{o}.

Abnormal palmar creases

The Simian transitional type I crease was significantly more in both hands of male treatment non-responders than in treatment responders. The Sydney line was significantly more in both hands of female treatment non-responders than in treatment responders.

t axial triradii position

The commonest position in treatment responders and non-responders was the t position of the axial triradius. The t' position of the triradius was seen more in both hands of male non-responders.

There is good correlation in the ridge counts of the digits I to V in both male responders and non-responders. There is good correlation in the ridge counts of the digits I to V in female responders and in digits I, III and V of female non-responders. There is no significant correlation in the ridge counts of digits II and IV in female non-responders indicating asymmetry, which is a feature of schizophrenia. There is significant correlation in the atd angles in treatment responders and non-responders (males and females). In the a-b ridge count, there is no significant correlation in treatment responders and non-responders (males and females), indicating asymmetry.

CONCLUSION

In the present work, various dermatoglyphic parameters on 144 schizophrenic patients who were responsive to treatment and 44 schizophrenic patients not responsive to the routine treatment were measured. The significant findings were as follows:

1) Increase in pattern frequency in the third interdigital area (I_3) of the left hand in treatment non-responders.
2) Increase in pattern frequency in the fourth interdigital area (I_4) of the right hand in treatment responders.
3) Decrease in the atd angle in the left hand of treatment non-responders.
4) Increase in the frequency of the Simian transitional type-1 crease in both hands of male non-responders.
5) Increase in the frequency of the Sydney line in both hands of female non-responders.
6) The t' position of the axial triradius is the most common position in both hands of male non-responders.
7) There is no significant correlation in the ridge counts of the second and fourth digits of female non-responders.
8) There is no significant correlation in the a-b ridge counts in male and female non-responders.

Thus, with the help of these parameters a schizophrenic patient likely to develop treatment resistance can probably be detected.

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