

VENTRICULAR HYPERTROPHY  
AND STRAIN

GRAND ROUNDS,  
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THE ELECTROCARDIOGRAPH MAY BE CLINICALLY HELPFUL ALONG 3 MAIN LINES, LISTED IN ORDER OF IMPORTANCE:

1. IT DISTINGUISHES, USUALLY IF NOT ALWAYS, BETWEEN TYPES OF CARDIAC ARRHYTHMIAS.
2. IT PROVIDES REASONABLY RELIABLE INFORMATION CONCERNING VENTRICULAR OVERLOAD, USUALLY DISTINGUISHING BETWEEN RIGHT AND LEFT VENTRICULAR INVOLVEMENT.
3. IT FURNISHES DIAGNOSTIC INFORMATION CONCERNING ACUTE MYOCARDIAL DAMAGE, ESPECIALLY IF USED SERIALY.
4. IT YIELDS INFORMATION OF QUESTIONABLE VALIDITY CONCERNING CHRONIC MYOCARDIAL DISEASE.

THE SECOND TOPIC IS OF GREAT CLINICAL IMPORTANCE AND IS A FOCAL POINT ABOUT WHICH A GREAT DEAL OF CONFUSION REVOLVES.

IT WAS EINTHOVEN HIMSELF (1906) WHO ORIGINALLY SET UP ELECTROCARDIOGRAPHIC CRITERIONS FOR LEFT AND RIGHT AXIS DEVIATION. HE BUILT THE FIRST STRING GALVANOMETER IN 1903 AND, ALONG WITH FAHR AND DEWAART, PUBLISHED THE EINTHOVEN TRIANGLE CONCEPT IN 1913. THIS VERY BASIC ARTICLE WAS TRANSLATED IN 1950 BY HOFF AND SEKELY; FROM IT COME ALL OUR CONCEPTS CONCERNING DETERMINATION OF ELECTRICAL POSITION AND VENTRICULAR PREPONDERANCE, AS WELL AS THE BASIC THEORY UNDERLYING VECTORCARDIOGRAPHY. THE AUTHORS POINT OUT VERY CLEARLY THAT ECG CHANGES DUE TO CHANGE IN POSITION OF THE HEART MAY RESEMBLE THOSE DUE TO VENTRICULAR HYPERTROPHY. THE MEAN QRS VECTOR (IN THE FRONTAL PLANE) IN THE NORMAL SUBJECT, ACCORDING TO EINTHOVEN, ET AL, WAS FROM  $+40$  TO  $+90$  DEGREES, WHILE IN "SEVERAL CASES OF LEFT VENTRICULAR HYPERTROPHY" IT WAS MARKEDLY NEGATIVE (UP TO  $-40$  DEGREES). PRESENT DAY ELECTROCARDIOGRAPHERS MIGHT CONSIDER THE NORMAL RANGE AS GIVEN BY EINTHOVEN A BIT TOO BROAD (IN ADULTS) AND A QRS AXIS OF  $-40$  DEGREES WOULD USUALLY BE CONSIDERED TO REPRESENT EXTREME LEFT AXIS DEVIATION (AND PROBABLY HYPERTROPHY). THE METHOD OF UTILIZING A TRIANGULAR FIGURE FOR THE GEOMETRIC CONSTRUCTION OF A TRUE ELECTRICAL VECTOR, POSSESSING DIRECTION, SIZE AND SENSE, IS EINTHOVEN'S. IT PROVIDED A BASIS FOR THE LOGICAL DEVELOPMENT OF ELECTROCARDIOGRAPHY ALONG VECTORIAL LINES; INSTEAD OF THIS, HOWEVER, THE TECHNIQUE CAME INTO CLINICAL USE AS AN EMPIRICAL TOOL IN WHICH STATE IT STILL REMAINS. DETERMINATION OF MEAN QRS, ST, AND T AXES IS SELDOM CARRIED OUT CLINICALLY, ALTHOUGH THE PROCESS IS NOT DIFFICULT, WHETHER THE ORIGINAL EINTHOVEN TRIANGLE OR THE MODIFIED TRAXIAL SYSTEM OF BAYLEY IS USED.

FOLLOWING EINTHOVEN, LEWIS AND HIS COLLABORATORS IN ENGLAND CAME POWERFULLY ON THE SCENE. IN 1914, LEWIS NOTED THE FUNDAMENTAL FACT THAT STRONG RIGHT-SIDED PATTERNS ARE NORMALLY PRESENT IN THE NEWBORN

AND DO NOT BECOME MODIFIED TOWARD THE ADULT PATTERN FOR SEVERAL MONTHS. HE ALSO NOTED THAT SOME ADULTS WITH LEFT VENTRICULAR OVERLOAD DO NOT SHOW CLEARCUT LEFT-SIDED ECG PATTERNS BUT THOUGHT THAT THE DIFFERENCE MIGHT BE MORE APPARENT THAN REAL. IT WOULD SEEM THAT LEWIS, AND CERTAIN AUTHORS WHO FOLLOWED HIM (COTTON, 1917), WERE EMPIRICALLY OBSERVING THE SIGNS OF AXIS DEVIATION IN THE STANDARD LIMB LEADS AND DID NOT, INITIALLY, DISTINGUISH BETWEEN SIMPLE AXIS DEVIATION AND A MORE SEVERE CHANGE. LEWIS DID, HOWEVER, MAKE REFERENCE TO THE RELATION BETWEEN INCREASED VOLTAGE AND VENTRICULAR HYPERTROPHY. VALUABLE THOUGH LEWIS' CONTRIBUTIONS UNDOUBTEDLY WERE, HE AND HIS PUPILS WERE LARGELY RESPONSIBLE FOR STEERING ELECTROCARDIOGRAPHY AWAY FROM THE SOUND LINES INDICATED BY EINTHOVEN AND COLLEAGUES IN 1913. PATTERN ELECTROCARDIOGRAPHY HAD ITS ORIGIN IN THE WRITINGS OF LEWIS AND OTHERS, AND SO GREAT WAS LEWIS' INFLUENCE THAT ANY ATTEMPT TO RETURN THE FIELD TO ITS BASICALLY SCIENTIFIC NATURE IS STILL LOOKED ON WITH SUSPICION. THE TOOL IS CAPABLE OF BEING USED AS A QUANTITATIVE AID TO DIAGNOSIS, WELL PROTECTED FROM THE FLABBINESS THAT IS PART AND PARCEL OF ANY TECHNIQUE WHICH IS OPEN TO SUBJECTIVE, NON-QUANTITATIVE INTERPRETATION. LEWIS AND THOSE WHO IMMEDIATELY FOLLOWED HIM EXPLOITED THIS NEW TWENTIETH CENTURY METHOD ALONG THE LINES LAID DOWN BY EIGHTEENTH CENTURY EMPIRICISTS AND THEIR EXPLOITATION WAS VERY WELL RECEIVED. WHITE AND BOCK (1918) IN FACT UTILIZED A MODIFIED LEWIS EMPIRICAL FORMULATION FOR THE RECOGNITION OF VENTRICULAR HYPERTROPHY. THE DEVICE SIMPLY ADDS THE AMPLITUDE OF  $R_1$  AND  $S_3$  (IGNORING THEIR NATURAL SIGNS), THEN SUBTRACTS THE SUM OF  $S_1$  AND  $R_3$  TO OBTAIN AN INDEX. A HIGH INDEX INDICATES A LEFT PATTERN, POSSIBLY LEFT VENTRICULAR HYPERTROPHY. FAIRLY HIGH VALUES IN SHORT, FAT PEOPLE INDICATE A HORIZONTAL HEART.

OTHER CONTRIBUTIONS WERE MADE BY CARTER AND GREENE (1919) AND BY FAHR (1920). THE LATTER MADE THE SIGNIFICANT OBSERVATION THAT IN MANY PATIENTS WITH HIGH VOLTAGE R WAVES IN LEAD I AND VERY DEEP S WAVES IN LEAD III, THE T WAVE IN LEAD I WAS OFTEN INVERTED. THIS IS A VERY EARLY, IF NOT THE FIRST, INTIMATION OF AN ELECTROCARDIOGRAPHIC PATTERN THAT GOES BEYOND SIMPLE AXIS DEVIATION MORPHOLOGICALLY AND THAT HAS A MORE SERIOUS CLINICAL IMPLICATION. FAHR, INCIDENTALLY, USED SIMPLE VECTORIAL ANALYSIS AND WAS AMONG THOSE WHO BROUGHT ORDER INTO CHAOS WITH REGARD TO THE DIAGNOSIS OF BUNDLE BRANCH BLOCK. HE WAS EMPHATIC (1921) IN OBSERVING THAT THE ELECTROCARDIOGRAM "GIVES NO DIRECT ANSWER TO THE QUESTION OF THE CONTRACTILE EFFICIENCY OF THE HEART MUSCLE."

THROUGH THE 1920'S AND 1930'S, A CONCEPT WAS GRADUALLY EVOLVED, THE END RESULT OF WHICH WAS THE ECG PATTERN WE NOW SOMETIMES REFER TO AS VENTRICULAR STRAIN. WILLIUS (1922) RELATED NEGATIVITY OF THE T WAVE IN LIMB LEAD I TO MORTALITY AND, IN THE PROCESS, CONFIRMED THE OBSERVATION ORIGINALLY MADE BY CARTER AND GREEN (LAD AND INVERSION OF  $T_I$  IN ASSOCIATION WITH ARTERIAL HYPERTENSION). LUTEN AND GROVE (1928) OBSERVED

WHAT THEY THOUGHT WAS THE SAME PATTERN BUT CONSIDERED THAT IT (LAD, QRS OF NORMAL DURATION, INVERTED T<sub>1</sub> AND UPRIGHT T<sub>3</sub>) INDICATED THE PRESENCE OF CORONARY DISEASE, NOT ARTERIAL HYPERTENSION. BARNES AND WHITTEN (1929) AGAIN EMPHASIZED THE ASSOCIATION OF NEGATIVITY OF T<sub>1</sub> WITH HYPERTENSION AND OTHER TYPES OF DISEASE PRODUCING LEFT VENTRICULAR OVERLOAD. THEY SEEM TO HAVE BEEN THE FIRST WHO ACTUALLY USED THE TERM VENTRICULAR STRAIN. IT WAS RYKERT AND HEPBURN (1935), HOWEVER, WHO COMPLETED THE PICTURE BY ADDING DEVIATION OF THE ST SEGMENT TO THE OTHER ELECTROCARDIOGRAPHIC FEATURES THAT HAD BEEN PREVIOUSLY ASSOCIATED WITH VENTRICULAR HYPERTROPHY AND STRAIN OR BOTH. THEIR PAPER IS IMPORTANT IN THAT IT ASSOCIATES THE FOLLOWING WITH MARKED LEFT VENTRICULAR DOMINANCE:

1. QRS<sub>1</sub> NORMAL IN DURATION BUT INCREASED IN AMPLITUDE;
2. LAD;
3. INVERTED T<sub>1</sub>, UPRIGHT T<sub>2</sub>;
4. DEPRESSED ST<sub>1</sub>, ELEVATED ST<sub>3</sub>.

THEY NOTED THAT THE ST SEGMENT DISPLACEMENT IS NOT USUALLY AS MARKED AS IN MYOCARDIAL INFARCTION AND STRESSED THE RELATIVE SPECIFICITY OF THE MORPHOLOGIC COMBINATION.

MEANWHILE, WILSON AND COLLEAGUES HAD BEGUN STUDIES ON WHAT WE NOW CALL THE UNIPOLAR ELECTRODE. WITH MACLEOD AND BARKER, WILSON DEFINED THE INTRINSICOID DEFLECTION (1930) AND PROCEEDED (1932) TO LAY DOWN THE TECHNIQUE AND SIGNIFICANCE OF UNIPOLAR ELECTROCARDIOGRAPHY (WILSON, JOHNSTON, MACLEOD AND BARKER, 1934). IN ACTUAL FACT, WILSON'S CONTRIBUTIONS REINSTATED THE VECTOR CONCEPT AND GAVE IT ANOTHER DIMENSION. AT THE SAME TIME, THE AUTHORS RECOGNIZED THAT ELECTROCARDIOGRAPHY HAD DEVELOPED ALONG PURELY EMPIRICAL LINES AND, FOR THIS REASON, HAD TO PRESERVE THE EMPIRICAL APPROACH IN THEIR LATER DESCRIPTIONS OF UNIPOLAR ELECTROCARDIOGRAPHIC PATTERNS. IN THEIR FIRST FULL-LENGTH PAPER ON UNIPOLAR ELECTROCARDIOGRAPHY (1934) THEY DESCRIBE THE CHANGES PRODUCED BY AXIS DEVIATION ON UNIPOLAR LIMB LEADS (ALTHOUGH THE POLARITY OF THEIR INSTRUMENT WAS THE REVERSE OF THAT NOW IN USE). SUBSEQUENTLY, THEY PUBLISHED VECTORGRAPHIC LOOPS ILLUSTRATING CHANGES INDUCED BY VENTRICULAR HYPERTROPHY (1938).

THE GRADUAL ACCEPTANCE OF PRECORDIAL LEADS AND, SOMEWHAT LATER, OF UNIPOLAR LIMB AND PRECORDIAL LEADS PRODUCED FURTHER DEFINITIONS OF HYPERTROPHY AND STRAIN PATTERNS. SIMPLE LEFT AXIS DEVIATION WAS NOW KNOWN TO BE OF LITTLE SIGNIFICANCE BY ITSELF. THE SEQUENCE LEADING FROM LAD, THROUGH LV STRAIN AND HYPERTROPHY TO LBBB WAS DESCRIBED IN 1934 BY GUBNER AND UNGELEIDER. TO THEM, THE OLD LEWIS CRITERIONS

( $R_1 + S_3 = 2.5$  mv., or more) WERE ADEQUATE FOR THE RECOGNITION OF LEFT VENTRICULAR HYPERTROPHY. THEY ALSO THOUGHT THAT THE SAME INTERPRETATION WAS INDICATED IF  $R_1$  WAS GREATER THAN 1.6 mv., OR  $S_3$  DEEPER THAN -1.6 mv. WHEN  $ST_1$  BECAME ELEVATED AND  $T_1$  INVERTED (WITH RECIPROCAL CHANGES IN LL III), THEY CONSIDERED LV STRAIN TO BE PRESENT BUT THOUGHT THAT ST-T CHANGES WERE LESS SPECIFIC THAN HIGH VOLTAGE  $R_1$  OR  $S_3$ . THEY ATTRIBUTED THE INCREASED VOLTAGE TO INCREASE IN LV MYOCARDIAL MASS AND ST-T CHANGES TO ISCHEMIA OF THE SUBENDOCARDIAL LEFT VENTRICULAR MYOCARDIUM. IF THE PROCESS CONTINUED LONG ENOUGH, THEY BELIEVED, SUBENDOCARDIAL FIBROSIS AND INTRAVENTRICULAR CONDUCTION DEFECTS MIGHT APPEAR. THEIRS WAS THE FIRST SYSTEMATIC EXPOSITION OF THE POSSIBILITY THAT LAD MIGHT BE REPLACED IN THE COURSE OF PROGRESSIVE LEFT VENTRICULAR OVERLOAD BY THE HYPERTROPHY (OR STRAIN) PATTERN AND THAT THE END RESULT MIGHT BE BUNDLE BRANCH BLOCK. KAPLAN AND KATZ (1941) MADE A FURTHER CONTRIBUTION BY EMPHASIZING THAT THE CHANGES IN ST-T IN LIMB LEAD I, AND NOT AXIS DEVIATION, WERE FUNDAMENTAL TO THE DIAGNOSIS OF LEFT VENTRICULAR STRAIN. THEY NOTED ABSENCE OF LAD ("CONCORDANT LVS") IN 19 PER CENT OF THEIR PROVED CASES. ABOUT THE SAME TIME, ROBB AND ROBB (1942) MADE THE FIRST PHYSIOLOGIC STUDY OF LVS (IN ANIMALS) AND SHOWED THAT WHEN THE LEFT VENTRICLE IS ACUTELY OVERLOADED, THE AMPLITUDE OF  $R_1$  INCREASES FIRST; THEN ST SEGMENTS TEND TO RISE BRIEFLY BUT AS PRESSURE IS FURTHER INCREASED,  $ST_1$  FALLS. THESE CHANGES, THEY INDICATED, CANNOT BE DUE TO HYPERTROPHY AND ARE RAPIDLY REVERSIBLE.

WITH THE ADVENT OF UNIPOLAR ELECTROCARDIOGRAPHY, AND ESPECIALLY WITH THE FANFARE SURROUNDING THE INTRODUCTION OF THE GOLDBERGER AUGMENTED UNIPOLAR EXTREMITY LEADS, AN OLDER POINT OF VIEW AGAIN RECEIVED EMPHASIS. SPECIFIC ELECTRODES WERE CONSIDERED TO "LOOK AT" SPECIFIC PORTIONS OF THE VENTRICLE (EPICARDIAL OR CAVITY SURFACES). GOLDBERGER (1944) REDEFINED THE ELECTROCARDIOGRAPHIC CRITERIONS FOR LEFT VENTRICULAR HYPERTROPHY BY NOTING THAT THE LEADS THAT FACE THE HYPERTROPHIED LV EPICARDIUM (AVL,  $V_4-6$ ) SHOW HIGH R WAVES, WHILE  $V_1-2$  AND AVR, SINCE THEY FACE THE LV CAVITY, SHOW DOWNWARD DEFLECTIONS. ST AND T CHANGES ARE USUALLY OPPOSITE TO QRS. RIGHT VENTRICULAR HYPERTROPHY WAS SAID TO SHOW ROUGHLY OPPOSITE CHANGES. GOLDBERGER'S MAIN PURPOSE WAS TO PROVIDE "A NONMATHEMATICAL APPROACH TO THE PROBLEM OF AXIS DEVIATION..."\* FURTHER DESCRIPTIONS, LARGELY ALONG PATTERN LINES, WERE PROVIDED BY MYERS AND ASSOCIATES (1948, 1950). THEY EMPHASIZED THE USEFULNESS OF

\* WHICH IS ACTUALLY NOT VERY SOUND. DEFLECTIONS AND THEIR MAGNITUDES ARE DETERMINED BY THE AXIS OF A PARTICULAR LEAD IN RELATION TO AN INSTANTANEOUS VECTOR. ALSO, DUCHOSAL SHOWED THAT LEADS TAKEN ON EXACTLY OPPOSITE SIDES OF THE CHEST SHOW EXACTLY REVERSE CONFIGURATIONS, SOMETHING WHICH DOES NOT FIT THE NOTION THAT A SPECIFIC ELECTRODE "LOOKS AT" A SPECIFIC PART OF THE HEART.



DELAY IN ONSET OF THE INTRINSICOID DEFLECTION (IN UNIPOLAR PRECORDIAL LEADS) FOR THE DIAGNOSIS OF VENTRICULAR HYPERTROPHY. IN THE CASE OF LVH, MYERS FOUND (1950) THAT THE PEAKS OF THE R WAVE WERE DELAYED IN  $V_{4-6}$  (.05 TO .07 SEC.), AND THAT THE DESCENDING LIMB OF THE S WAVE IN  $V_{1-4}$  WAS SLOW IN REACHING ITS NADIR. DEPRESSED ST AND INVERSION OF T IN  $V_{4-6}$  WERE ALSO CHARACTERISTIC, AS WAS A SLURRING OF THE R UPSTROKE IN PRECORDIAL LEADS NEAR THE TRANSITION ZONE (USUALLY  $V_3$  OR  $V_4$ ).

THESE, THEN ARE THE CRITERIONS DEVELOPED IN PATTERN ELECTROCARDIOGRAPHY FOR THE DIAGNOSIS OF VENTRICULAR DOMINANCE. IN VERY GENERAL TERMS, SIMPLE AXIS DEVIATION IS NOT, OF ITSELF, ABNORMAL. THE DIAGNOSIS OF VENTRICULAR HYPERTROPHY IS USUALLY HELD TO REQUIRE INCREASED VOLTAGE IN APPROPRIATE LEADS. FOR LVH,  $R_1$  AND  $S_1$  ARE INCREASED, AS ARE R WAVES IN  $V_{4-6}$ . QRS IS NOT PROLONGED BUT THE INTRINSICOID DEFLECTION MAY BE DELAYED. FOR RVH,  $S_1$  AND  $R_3$ , AS WELL AS  $R_1$  IN  $V_{1-4}$  ARE ORDINARILY ABNORMALLY LARGE. THE DIAGNOSIS OF VENTRICULAR STRAIN REQUIRES THE PRESENCE OF ST AND T WAVE CHANGES OF APPROPRIATE NATURE. THEY ARE OFTEN, BUT NOT ALWAYS, ASSOCIATED WITH THE ELECTROCARDIOGRAPHIC PATTERNS OF HYPERTROPHY.

BUNDLE BRANCH BLOCK REQUIRES THAT QRS BE PROLONGED (0.10 SEC. OR GREATER) AND IS BEYOND THE SCOPE OF THIS DISCUSSION. IN PASSING, ONE SHOULD NOTE THAT BBB IS NOT NECESSARILY A SIGN OF VENTRICULAR HYPERTROPHY. IT SOMETIMES REPRESENTS A VERY LATE STAGE OF VENTRICULAR HYPERTROPHY, BUT MORE OFTEN IS ASSOCIATED WITH CORONARY DISEASE. A CAREFULLY CONTROLLED STUDY OF PROLONGATION OF QRS IN PATIENTS WITH AND WITHOUT LVH WAS DONE BY GRANT AND DODGE (1956). THEY SHOW THAT PERI-INFARCTION BLOCK AND LVH MAY PRODUCE VERY SIMILAR LENGTHENING OF QRS WITHOUT DISTURBING ITS .03 OR .04 SECOND VECTOR COMPONENTS. TRUE LBBB, ON THE OTHER HAND, ALWAYS AFFECTS THESE EARLIER VECTOR COMPONENTS OF QRS.

THE ONLY SERIOUS ATTEMPT TO INTRODUCE SPATIAL (3 DIMENSIONAL) VECTOR CONCEPTS INTO CLINICAL PRACTICE IS THAT OF GRANT (1951, 1957) WHOSE METHOD STEMS ENTIRELY FROM WILSON'S CONCEPTS. USING LIMB AND UNIPOLAR PRECORDIAL LEADS, GRANT PLOTS MEAN QRS, ST, AND T VECTORS IN WHAT AMOUNTS TO 3 PLANES, (FRONTAL, SAGGITAL, AND HORIZONTAL). VENTRICULAR HYPERTROPHY ALTERS THE DIRECTIONS OF THE VECTORS IN A CHARACTERISTIC MANNER AND THIS TYPE OF ANALYSIS PERMITS IDENTIFICATIONS THAT ARE NOT POSSIBLE BY USUAL TECHNIQUES. IN THE CASE OF LVH, THE MEAN QRS VECTOR IS INCREASED IN AMPLITUDE BUT MAY BE NORMAL IN DIRECTION (ESPECIALLY IN YOUNG ADULTS), OR MAY BE DIRECTED HORIZONTALLY. HYPERTROPHY IS SAID TO AFFECT THE EPICARDIAL LAYERS OF THE MYOCARDIUM RATHER THAN THE INTERNAL FIBERS. SINCE THE MIDDLE AND LATER PORTIONS OF THE QRS COMPLEX ARE GENERATED WITHIN THE EPICARDIAL LAYERS, INCREASED AMPLITUDE AND DELAY IN THE INTRINSICOID DEFLECTIONS NATURALLY RESULT. SINCE THE QRS MEAN VECTOR IS

OFTEN DIRECTED POSTERIORLY, AS THE LV HYPERTROPHIES, INCREASED AMPLITUDE OF THE R WAVE MAY BE MORE APPARENT IN THE LATERAL PRECORDIAL LEADS THAN IN LL I. THIS, PLUS THE FACT THAT AMPLITUDE VARIES INVERSELY AS THE SQUARE OF THE DISTANCE OF THE ELECTRODE FROM THE CURRENT SOURCE, MAY LEAD TO DIAGNOSTIC ERROR. VERY THIN-CHESTED PEOPLE OFTEN SHOW R AMPLITUDES THAT WOULD INDICATE VENTRICULAR HYPERTROPHY IF JUDGED BY THE OLD LEWIS-WHITE CRITERIONS. A VERY FAT-CHESTED PERSON, ON THE OTHER HAND, MAY SHOW NORMAL R WAVES IN SPITE OF CONSIDERABLE VENTRICULAR HYPERTROPHY. VECTOR ANALYSIS MAY HELP IN AVOIDING SUCH ERRORS. THE MOST IMPORTANT VECTORGRAPHIC CRITERION FOR VENTRICULAR DOMINANCE, AND VENTRICULAR STRAIN IN PARTICULAR, IS THE ANGLE FORMED BY THE QRS VECTOR, ON THE ONE HAND, AND THE ST-T VECTORS ON THE OTHER. NORMALLY, MEAN QRS AND T VECTORS POINT IN THE SAME GENERAL DIRECTION; IN LVS, THEY POINT IN NEARLY OPPOSITE DIRECTIONS, FORMING AN ANGLE OF ABOUT 180 DEGREES. THE RELATIONSHIP REQUIRES 3-DIMENSIONAL REPRESENTATION FOR ITS RECOGNITION BUT IS QUITE SPECIFIC. IT SERVES TO DISTINGUISH THE LVS PATTERNS FROM THOSE DUE TO DIGITALIS EFFECT, CORONARY INVOLVEMENT, AND OTHER ABNORMAL STATES. DIGITALIS, FOR EXAMPLE, MAY SHIFT THE ST VECTOR NEARLY 180 DEGREES BUT DOES NOT ALTER THE T VECTOR UNTIL QUITE LATE. PERICARDITIS DOES JUST THE OPPOSITE. SUBENDOCARDIAL OR SUBEPICARDIAL DAMAGE ARE ALSO SEPARABLE FROM STRAIN PATTERNS BY MEANS OF THE VECTOR APPROACH.

SUMMARY

A.

I	II	III	IV
(A) AXIS DEV. ALONE	(A) SIMPLE HYPER- TROPY PATTERNS	(A) STRAIN, WITH OR WITHOUT HYPERTROPHY	(A) BBB*
	(B) SIMPLE HYPER- TROPY WITH SLIGHT ST-T CHANGE	(B) STRAIN OR HYPER- TROPY WITH SLIGHT PROLONGATION OF QRS	
	(C) REVERSIBLE (AND REVERSED) "STRAIN", WITH OR WITHOUT IN- CREASED AMPLITUDE		

I. NO CLINICAL SIGNIFICANCE.

II. VENTRICULAR OVERLOAD.

III. SEVERE VENTRICULAR OVERLOAD.

IV. SAME AS III, WITH SUBENDOCARDIAL DAMAGE.

\* NOT TRUE BBB, WHICH IS EITHER CONGENITAL OR DUE TO CORONARY DISEASE.

B. THE HYPERTROPHY AND STRAIN CONCEPTS SERVE VERY USEFUL CLINICAL CONCEPTS BUT NEED TO BE DEFINED MORE UNIFORMLY.

C. THE VECTOR APPROACH IS THE BEST MEANS OF PROPERLY IDENTIFYING QUESTIONABLE CASES.

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