Gallop Rhythm of the Heart

II. Quadruple Rhythm and its Relation to Summation and Augmented Gallops

By Joseph Grayzel, M.D.

The 2 fundamental types of gallop are ventricular gallop and atrial gallop. Adequate cardiac acceleration modifies these gallops and may produce a summation gallop, an augmented ventricular gallop, or an augmented atrial gallop. The summation and augmented gallops were examined and their relation to the 2 fundamental gallops was quantitated. The cardiac rate at which summation and augmentation occur is unique. A graph and 2 corresponding equations were derived which relate the summation cardiac rate and corresponding cycle length to familiar electrocardiographic and phonocardiographic intervals.

Gallop rhythm has been defined as a "mechanical hemodynamic event associated with a relatively rapid rate of ventricular filling and characterized by a ventricular bulge and a low-frequency sound."1 From this definition it follows that the cardiac gallop is a diastolic event. Of the 5 divisions of diastole—protodiastole of Wiggers, isometric ventricular relaxation, rapid ventricular filling, slow ventricular filling, and atrial contraction—a relatively rapid rate of ventricular filling occurs during 2 periods, the rapid-filling phase, which follows immediately upon opening of the atrioventricular valve, and the atrial phase, which follows contraction of the upper chamber. Two corresponding types of gallop exist. These are the rapid-filling (or ventricular) gallop and the atrial gallop, respectively. Both types of gallop are ventricular phenomena and each may be generated in either ventricle. Therefore, it is desirable to specify whether a gallop originates from the right or left side of the heart.

Gallop may occur at any heart rate. Associated mechanical aspects of the gallop have been recorded with the electrokymogram,2 slit roentgenkymogram,3 ballistocardiogram,4,5 and apex cardiogram.1

Ventricular gallop appears indicative of diastolic overload. It occurs with an abnormal relation between the rate of rapid filling and the ventricle's ability to accommodate its increasing diastolic volume. The wave of rapid left ventricular filling is increased in mitral insufficiency, in which left atrial volume is large, the atrial pressure is high, and mitral stenosis is not of a degree to restrain the rate of flow from atrium to ventricle.

In aortic insufficiency the regurgitant blood stream augments left ventricular filling and the total rapid-filling wave exceeds that normally present.

Left-to-right shunts at the ventricular level or between the great vessels result in increased left ventricular diastolic filling and stroke volume. Examples are interventricular septal defect, patent ductus arteriosus, and aortic-pulmonary window.

Interatrial septal defect increases diastolic filling of the right ventricle and rapid-filling gallop of the right ventricle may occur.

More common than ventricular gallop due to an increased filling wave is ventricular gallop due to altered accommodation during the volume changes of diastole. The most frequent cause is heart failure, in which the ventricle is dilated and myocardial tone is poor.

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Left ventricular gallop occurs 0.15 second after the onset of the second heart sound.\textsuperscript{1, 4, 6, 7} An exception is mitral insufficiency, in which this gallop begins 0.10 second after the second heart sound\textsuperscript{1} due to earlier opening of the mitral valve when left atrial pressure is elevated, ending a shortened period of isometric ventricular relaxation.

Atrial gallop appears indicative of systolic ventricular overload. It is generated on the left side of the heart in essential hypertension and aortic stenosis, and on the right side in pulmonary hypertension of varied etiology and in pulmonic stenosis.

When atrial gallop is present in essential hypertension, there is invariably evidence of left ventricular hypertrophy, either from physical examination, roentgenograms, or the electrocardiogram.\textsuperscript{1, 8} The atrial gallop will often persist when the blood pressure in a known hypertensive person is within normal range. This observation suggests that the ventricular changes, rather than the elevated blood pressure per se, are essential for the production of this presystolic gallop. In the known hypertensive person the presence of an atrial gallop warrants a diagnosis of hypertensive heart disease.

The interval from the onset of the P wave of the electrocardiogram to the left atrial gallop is 0.14 second.\textsuperscript{1, 6, 9} The inaudible vibrations of mechanical atrial contraction occur earlier than the atrial gallop. Many of the atrial sounds heard in various degrees of heart block occur later than true atrial gallop.

Quadruple rhythm denotes the presence of 4 heart sounds. The special case of concern here is quadruple rhythm due to the occurrence of both left ventricular and left atrial gallops in addition to the normal first and second heart sounds.

As the heart rate increases, the cycle length decreases, principally at the expense of diastole. More specifically, the period of slow ventricular filling is shortened and even totally eliminated with tachycardia of sufficient degree. At this point a slight additional increase in rate results in superposition and summation of the rapid-filling phase and atrial contraction (summation phenomenon). When both the rapid-filling and atrial gallops are present, they are superimposed as summation occurs. The resulting single, intense gallop is called summation gallop. The single sound is usually much louder than would be obtained from simple addition of the 2 component sounds; this gives the impression that the intensities are multiplied rather than summed as the name implies.

When the summation gallop occurs, mechanical and auscultatory aspects of the 2 component gallops appear summated. There is summation of the respective ventricular thrusting forces as well as summation of the sounds.

A rapid ventricular filling gallop is greatly intensified when a previously silent phase of atrial contraction is superimposed upon it by an increased heart rate. The single ventricular gallop intensified by this mechanism is augmented both mechanically and acoustically, and is called augmented ventricular gallop. Similarly, an atrial gallop superimposed upon a silent period of rapid ventricular filling is intensified and is called augmented atrial gallop. Thus, the mechanism of the augmented gallop and the true summation gallop is similar in that superposition of the rapid ventricular filling period and the period of atrial contraction is essential to both.

The intensity of an augmented gallop and the loudness of the true summation gallop make these easily audible and probably account for the repeated statement that a gallop is only heard during tachycardia.

A single loud gallop sound present during tachycardia may be either an augmented gallop or a true summation gallop. The distinction can be made only when the heart rate is slowed. The summation gallop will change to a quadruple rhythm at the slower rate. The augmented gallop will only decrease in intensity, the sound remaining single. If an augmented ventricular gallop is present, the sound will retain its relation to the
second heart sound and remain in early diastole. If an augmented atrial gallop is present, the sound will retain its relation to the P wave and remain presystolic.

**Method and Materials**

Phonocardiograms were recorded simultaneously with the electrocardiogram and apex cardiogram on a Sanborn Twin-beam at a paper speed of 75 mm. per second with vertical time lines at 0.04 second. The patients were in the supine position. The method for recording local precordial movements has been described previously. An upward deflection on the apex cardiogram represented a forward movement in the region of maximum cardiac pulsation.

Observations on 5 patients with gallop sounds and the pertinent clinical details are presented. Quadruple rhythm due to the presence in each cardiac cycle of both the rapid-left-ventricular-filling gallop and the left atrial gallop was recorded in 2 patients. The various time intervals associated with these gallops have been included among previous observations. An augmented gallop or a true summation gallop was recorded in 3 patients.

**Case Reports**

**Case 13**

E.C. was a 52-year-old Negro with a history of progressive exertional dyspnea for 2 years and recent orthopnea. On physical examination the blood pressure was 180/110, there was grade-II hypertensive retinopathy, and rales were present at both lung bases. The heart was moderately enlarged, and no murmurs were audible. Excursions at the point of maximum cardiac pulsation were complex: a prominent early diastolic bulge was present as well as a presystolic bulge, the latter giving the impression of a double systolic impulse. Upon auscultation the normal first and second heart sounds were easily identified. Two low-pitched sounds were present in diastole. The first occurred soon after the second heart sound and corresponded in timing to the early diastolic, left ventricular bulge. The second low-pitched sound occurred with the presystolic bulge.

A phonocardiogram (fig. 1) showed the low frequency (fundamental 25 c.p.s.) early diastolic sound occurring 0.16 second after the second heart sound. The presystolic sound was also of low frequency (fundamental 25 c.p.s.) and followed the P wave by 0.12 second. Both diastolic sounds occurred simultaneously with a prominent ventricular bulge on the apex cardiogram, indicating the presence of rapid-filling gallop and atrial gallop, respectively.

**Comment.** Atrial gallop of the left heart, which is associated with systolic overload of the left ventricle, was caused in this patient by essential hypertension. The electrocardiogram was normal. Rapid-filling gallop, which accompanies diastolic overload, in this case was the result of heart failure.

**Case 14**

W.O.H. was a 24-year-old white man who had experienced recurrent episodes of acute hemorrhagic glomerulonephritis since childhood. In 1954 hypertension and impaired renal function were present. In 1958 he was hospitalized for severe heart failure and terminal renal failure. At this time the blood pressure was 180/110. There was grade-III hypertensive vascular retinopathy. Rales were heard over both lung fields. The heart was enlarged to the anterior axillary line. No murmurs were audible. The cardiac pulsations were undulating in character and suggested myocardial disease. A relatively distinct forward movement could, however, be consistently discerned in early diastole and was more distinct than the systolic impulse. Coincident with this early diastolic bulge was a low-pitched sound, loudest near the apical region. A faint presystolic sound of low pitch was also heard at the apex, but was loudest in the left fourth interspace. The neck veins were distended, the liver was tender and enlarged, and there was 4-plus pitting edema of the legs and ankles.

The hematocrit value was 22 per cent and the blood urea nitrogen was 228 mg. per 100 ml. The electrocardiogram was within normal limits. A phonocardiogram recorded both diastolic sounds with fundamental frequencies of 35 and 50 c.p.s., respectively. The early diastolic sound was loudest near the apex (fig. 2B) and the presystolic sound was loudest in the left fourth interspace (fig. 2A). The apex cardiogram (fig. 2C) illustrated the undulating cardiac movement felt through the chest wall. However, a prominent early diastolic bulge appeared consistently, simultaneous with the early diastolic sound, and indicated the presence of a rapid-filling gallop. A frank presystolic bulge simultaneous with the atrial presystolic gallop sound was not demonstrable. Presumably, the presystolic undulating wave occurring with the sound was in part due to the gallop bulge.

**Comment.** The atrial presystolic gallop in this case reflected hypertensive heart disease resulting from hypertension of 4 years’ duration, secondary to renal disease. Left ventricular hypertrophy was confirmed at postmortem examination, although electrocardiographic evidence of hypertrophy was not present. The rapid-filling gallop reflected the
myocardial failure. It is commonly found, as in this case, that the atrial and ventricular gallop sounds are loudest in different areas of the precordium.

Case 15

L.L. was a 51-year-old obese Negro with known hypertension for 6 years and symptoms of congestive heart failure during the 3 years prior to this hospitalization for severe dyspnea, orthopnea, and massive edema.

The blood pressure was 192/130 with 12 mm.

of systolic alternation. The minute pulse rate was 107. The oculur fundi showed grade-II hypertensive vascular changes. There were basilar rales in both lungs. The heart was enlarged to the anterior axillary line. No murmurs were audible, but 3 heart sounds were present: the first sound was soft, and the second sound and the extra diastolic sound were of equal intensity, but the latter was of low pitch. The chest wall was obese and a diastolic ventricular bulge could not be detected. The neck veins were distended and there was 4-plus pitting edema of the ankles, legs, and lower thighs. An electrocardiogram showed left axis deviation but no evidence of hypertrophy.

A phonocardiogram (fig. 3) demonstrated the diastolic sound of low frequency (fundamental 50 cycles per second) which occurred 0.15 second after the second heart sound and 0.13 second after the P wave of lead II. A satisfactory apex cardiogram could not be recorded through the obese chest wall.

Comment. The diastolic gallop sound followed the second heart sound by the proper interval for a rapid-filling left ventricular gallop and followed the P wave by the proper interval for a left atrial gallop. It is justified to conclude that the summation phenomenon was present. Subsequently, with a minute cardiac rate of 90, quadruple rhythm was present due to the occurrence of the ventricular and atrial gallops separately with each cardiac cycle. Therefore, the single diastolic sound recorded at a heart rate of 107 represented a true summation gallop as opposed to an augmented gallop as defined above. Atrial gallop in this hypertensive patient indicated myocardial hypertrophy. Ventricular gallop was the result of diastolic ventricular overload secondary to heart failure.

Case 16

W.I. was a 32-year-old Negro who had been observed for 2½ years with congestive heart failure. The clinical impression was “idiopathic myocardial failure,” commonly seen in young Negroes. At this time the blood pressure was 120/70 and the minute pulse 108. Basilar rales were heard bilaterally. The heart was enlarged well to the
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anterior axillary line. A blowing grade-II apical systolic murmur was audible. A loud low-pitched diastolic sound was present and coincided with a relatively large forward movement of the region of maximum cardiac pulsation. The liver descended 1 fingerbreadth below the right costal margin and was tender. There was 1-plus ankle edema. An electrocardiogram was within normal limits.

A phonocardiogram (fig. 4A) demonstrated the galloping sound occurring 0.16 second after the second heart sound and 0.13 second after the P wave of lead II. The simultaneous ventricular bulge on the apex cardiogram was striking (fig. 4B).

Comment. The time interval from the second heart sound to the gallop was appropriate for a rapid-filling left ventricular gallop. The interval from the P wave to the gallop was appropriate for a left atrial gallop. Thus, the summation phenomenon is present. At a slower cardiac rate the gallop remained single, was of less intensity, and maintained its relation to the second heart sound. The record shown (fig. 4) is therefore, an example of an augmented ventricular gallop.

Case 17

J.H.S. was a 35-year-old white man who had been observed for 2 years with heart failure. The clinical impression was "idiopathic myocardial failure." There was never any evidence of hypertension or coronary artery sclerosis. The blood pressure was 105/90 and the minute pulse 104. The ocular fundi were unremarkable. Rales were heard at both lung bases. The heart was moderately enlarged. A very low pitched diastolic sound was audible and coincided with a prominent apical bulge. This galloping sound was the loudest of the 3 heart sounds. The first heart sound was particularly soft. The neck veins were slightly distended and there was 3-plus ankle edema. An electrocardiogram showed complete left bundle-branch block.

A phonocardiogram (fig. 5A) demonstrated a galloping sound of high intensity and low frequency (fundamental 45 c.p.s.), which occurred simultaneously with the sharp spike on the apex cardiogram (fig. 5B).

Comment. The simultaneous sharp ventricular bulge established the diastolic sound to be a gallop. It occurs 0.13 second after the second heart sound, which is slightly less than the mean value for this interval but still appropriate for a left ventricular gallop. The P wave was lost in the preceding T wave with a P-R interval of 0.20 to 0.22 second. The interval between the P wave and the gallop was in the range appropriate for a left atrial gallop. The great intensity of the gallop compared to the first and second heart sounds further supports the conclusion that the summation phenomenon was present. This patient was not observed at a slower cardiac rate and we are therefore unable to say whether the gallop was a true summation gallop or an augmented gallop.

DISCUSSION

Over the range of cardiac rates usually encountered the rapid-filling or ventricular galloping sound bears a constant relation to the second heart sound. The mean value for the interval between the second heart sound and the left ventricular gallop (2-vG interval) is 0.15 second. The ventricular gallop in mitral regurgitation is a special case and is not included in the calculation of this mean value.

The left atrial gallop follows the P wave (P-aG interval) by 0.14 second. When cardiac acceleration is sufficient to eliminate the period of slow ventricular filling and cause superposition of the ventricular and atrial gallop sounds, the true summation gallop results. Therefore, the true summation gallop should possess those timing features characteristic of its 2 component gallops. It should follow the second heart sound by 0.15 second and follow the P wave by 0.14 second. It becomes evident that the heart rate at which precise summation occurs is not a matter of chance but is uniquely determined by the different time intervals that comprise a single cardiac cycle.

Figure 6 illustrates a single auscultatory cardiac cycle from one first heart sound to the next. The second heart sound and the summation gallop are also indicated. Above
the auscultatory cycle is the simultaneous electrocardiogram showing the P wave and the QRS complex. The single cycle is divided into intervals to enable calculation of the cycle duration at which the summation gallop occurs. The duration of this cycle is the sum of its component subdivisions as constructed. We observe from figure 6 that

\[
\text{Summation cycle length (seconds/cycle)} = S + (2-\nu G) + (PR) - (P-aG) + (Q-1) \quad (1)
\]

This expression is accurate for any given case but is cumbersome for general use. It is simplified by substituting for some of the intervals their numerical mean values. The following values are employed: \(S = 0.29\) second. This is the interval between the first and the second heart sounds at a cardiac rate of 109. This rate lies in the middle range of rates at which summation occurs. \((2-\nu G) = 0.15\) second. \((P-aG) = 0.14\) second. These are the most accurate mean values to 2 significant figures for the left heart. The P-aG interval was determined in hypertensive heart disease, which is the most frequent cause of left atrial gallop. It may be a valid mean for other conditions of systolic overload, such as aortic stenosis, but such a statistical study is not available. \((Q-1) = 0.07\) second This is the mean value for this interval in hypertensive heart disease.\(^{1,10}\)

The P-R interval is left as a variable since its variations are random and occur over a wide range.

On substitution of the above values, equation 1 becomes

\[
\text{Summation cycle length (seconds/cycle)} = (0.29) + (0.15) + PR - (0.14) + (0.07)
\]

\[
\text{seconds/cycle} = 0.37 + PR \quad (2)
\]

Taking reciprocals on both sides of equation 2 yields

\[
\text{cycles/second} = \frac{1}{0.37 + PR}
\]

Multiplication by 60 gives minute heart rate

\[
\text{cycles/min.} = \frac{60}{0.37 + PR} \quad (3)
\]

Equation 3 provides a useful relation between the heart rate in beats per minute
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at which precise summation occurs (i.e., the summation rate) and the P-R interval in seconds. Table 1, column 2, lists corresponding values for the summation cardiac rate and the P-R interval, as calculated from equation 3.

The relation expressed by equations 2 and 3 is depicted graphically in figure 7. This graph was constructed by employing the mean values for the 2-vG, P-A-G, and Q-1 intervals. The horizontal width of the figure at any level represents the duration of a single cycle at the corresponding heart rate. The timing of the first, second, rapid-filling gallop, and atrial gallop sounds is indicated by the appropriate straight line. The time of occurrence of the atrial gallop depends upon the timing of the P wave and therefore will vary with the P-R interval. The point of intersection of the time lines representing the rapid-filling and atrial gallops is the point of precise or complete summation. The ordinate or horizontal level of this point gives the cycle duration or the cardiac rate at which summation occurs. The graph illustrates the dependence of the summation cardiac rate upon the P-R interval.

The summation rates for various P-R intervals, determined from the graph, are listed in table 1. At a P-R interval of 0.18 second the summation rate is 109 and is identical with that obtained from equation 3.

next. Cycle duration in seconds is represented on the vertical axis (ordinate) and appears on the right; the corresponding minute heart rate appears on the left. Distance along the horizontal axis (abscissa) represents time elapsed from the first heart sound that begins the cycle. The constant relation between the second heart sound and the rapid-filling gallop is represented by respective parallel lines 0.15 second apart. The constant relation between the P wave and the atrial gallop results in a constant interval from the atrial gallop to the ensuing first heart sound for a given P-R interval. The lines representing the time of atrial gallop for a given P-R interval and the ensuing first heart sound are parallel and the distance between them is PR — (P-A-G) + (Q-1) = PR — 0.14 + 0.07 = PR — 0.07 (in seconds). The intersection of the 2 time lines representing the 2 gallops is the point of precise summation. The ordinate of this point is the summation rate (left of figure) or the summation cycle length (right of figure).

Fig. 6 Top. Schematic representation of simultaneous electrocardiogram (upper tracing) and phonocardiogram (lower tracing) for a single auscultatory cycle in which the summation phenomenon occurs precisely. Then, the gallop sound (G) should possess the timing features of both a rapid-filling gallop and an atrial gallop. The single cycle is then divided into intervals to enable calculation of the cycle length in terms of known quantities. The P wave and the QRS complex are labeled as such. 1, first heart sound; 2, second heart sound; G, summation gallop or augmented gallop; S, duration of auscultatory systole; 2-vG, interval from the second heart sound to the rapid-ventricular-filling gallop; PR, electrical P-R (P-Q) interval; P-A-G, interval from onset of the P wave to the atrial gallop; Q-I, interval from onset of the QRS complex to the first heart sound.

Fig. 7 Bottom. Graphic representation of a single auscultatory cycle from one first heart sound to the
TABLE 1.—Summation Rate as a Function of the P-R Interval

<table>
<thead>
<tr>
<th>P-R interval (sec.)</th>
<th>Summation rate, beats per minute, calculated from</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Equation 3</td>
</tr>
<tr>
<td>0.14</td>
<td>118</td>
</tr>
<tr>
<td>0.16</td>
<td>113</td>
</tr>
<tr>
<td>0.18</td>
<td>109</td>
</tr>
<tr>
<td>0.20</td>
<td>105</td>
</tr>
<tr>
<td>0.22</td>
<td>102</td>
</tr>
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</table>

This precise agreement reflects the substitution in equation 1 of 0.29 second for $S$, the duration of auscultatory systole at a minute heart rate of 109. The graph of figure 7 takes account of the slight variation of systole with heart rate, and is more accurate to this degree than is equation 2 or 3. Nevertheless, in the range of normal P-R intervals the error of equation 3 resulting from the substitution of a constant value for $S$ is less than 2 per cent.

These calculations may be extended to include the augmented ventricular gallop and the augmented atrial gallop. When ventricular gallop alone is present, cardiac acceleration can superimpose this gallop on the time when atrial gallop would occur (i.e., 0.14 second after the onset of the P wave). The ventricular gallop is then augmented, as evidenced by the marked increase in sound intensity and often by the magnitude of the ventricular bulge. The same is true for an existing atrial gallop superimposed upon the appropriate portion of the rapid-filling period (i.e., 0.15 second after the onset of the second heart sound). Therefore, equations 2 and 3 and the graph are valid for the 2 types of augmented gallop as well as for the true summation gallop.

The fortuitous timing of a premature atrial contraction can momentarily superimpose the phase of atrial contraction upon the period of rapid-ventricular filling and thereby produce the summation phenomenon. When both ventricular and atrial gallops are present, and the premature P wave begins 0.14 second before the ventricular gallop, the 2 gallops are superimposed to produce a summation gallop. If only a ventricular gallop were originally present, the same premature atrial contraction will momentarily augment the existing ventricular gallop. For the abbreviated cycle which is ended by the premature atrial contraction equation 2 is valid and expresses the duration of the shortened cycle in terms of the P-R interval of the premature atrial systole, only if the summation phenomenon has occurred. Conversely, if the values for the P-R interval of the premature atrial systole and the length of the abbreviated cycle satisfy equation 2, then summation certainly has occurred. Reexamination of figure 6 provides a visual aid in understanding summation due to a premature atrial contraction. One must now imagine that the P wave and the QRS complex shown are those of the premature atrial contraction, which has encroached upon the normal diastolic period to produce summation.

Clinical Significance. The true summation gallop and the augmented gallops occur when the heart rate is sufficiently fast. The significance of the true summation gallop is that of its 2 components. The significance of the augmented gallop is that of the single gallop identified when the heart rate is slower. Summation and augmentation only reflect the more rapid heart rate.

Summary

Adequate cardiac acceleration will superimpose the phase of atrial contraction upon the period of rapid ventricular filling. The superposition of these 2 periods is called the summation phenomenon. The cardiac rate at which precise summation occurs and its corresponding cycle length are termed the summation rate and the summation cycle length, respectively.

Both the ventricular gallop and the atrial gallop may be present in each cardiac cycle, producing a quadruple rhythm. When cardiac acceleration is sufficient to produce the summation phenomenon, the 2 gallops are
superimposed to produce the true summation gallop.

When a ventricular or an atrial gallop alone is present the summation phenomenon will augment the intensity of the existing gallop. It is then called augmented ventricular gallop or augmented atrial gallop, respectively.

A single, loud gallop present during tachycardia may be either an augmented gallop or a true summation gallop. The distinction can be made only when the heart rate is slowed, at which time the true summation gallop will change to a quadruple rhythm while the augmented gallop remains a single sound of reduced intensity.

Examples of quadruple rhythm, summation gallop, and augmented gallop are shown.

Two equations were derived that express the summation cycle length or the summation rate, respectively, as a function of the P-R interval. A graphic representation of this relation was also constructed. Of the critical time intervals the P-R interval is the only random variable affecting the summation rate, and its range is the largest.

The equation for the summation cycle length is also valid when the summation is produced by a premature atrial contraction. The cycle in which summation occurs is the shortened cycle, prematurely ended. The duration of such a summation cycle is a function of the P-R interval of the premature atrial contraction, as expressed by the equation derived for the more usual case of summation resulting from cardiac acceleration.

**Summario in Interlingua**

Adequate acceleration cardiac impone le phase de contraction atrial super le periodo de rapide replenation ventricular. Le superimposition de iste duo periodos es appellate le phenomeno de summation. Le frequencia cardiac al qual un summation precise occurre e le correspondente longor de cyclo es designate, respectivemente, como le frequencia de summation e le longor de cyclo de summation.

Tanto le galopo ventricular como etiam le galopo atrial pote esser presente in un ciclo cardiac individual, con le resultante production de un rhythmio quadruple. Quando le acceleration cardiac es sufficientemente intense pro producer le phenomeno de summation, le duo galopos es superimponite le un al altere con le resultante production del ver galopo de summation.

Quando un galopo ventricular sol o un galopo atrial sol es presente, le phenomeno de summation augmenta le intensitate del galopo existente. Alora illo es appellate un augmentate galopo ventricular o, respectively, un augmentate galopo atrial.

Un sol e forte galopo que es presente durante tachycardia pote esser (1) un galopo augmentate o (2) un ver galopo de summation. Le distinction inter le duo pote esser facite solmente post retardar le frequencia cardiac, quando le ver galopo de summation se transforma in un rhythmio quadruple, durante que le galopo augmentate remane un sol sono de intensitate reduceite.

Es presentate exemplos de rhythmio quadruple, de galopo de summation, e de galopo augmentate.

Esseva derivate duo equationes que exprime, respectivemente, le longor de cyclo de summation e le frequentia de summation como functiones del intervallo P-R. Un representation graphic de iste relation esseva etiam construite. Inter le critic intervallos de tempore, le intervallo P-R es le sol variabile de hasardo que affice le frequentia de summation, e su gamma de valores possibile es le plus extense.

Le equation pro le longor de cyclo de summation remane valide quando le summation es producite per un prematur contraction atrial. Le cyclo in qu le summation occurre es le cyclo abbreviate con termination prematur. Le duration de un tal cyclo de summation es un function del intervallo P-R del contraction atrial prematur, exprimite per le equation que es derivate pro le caso plus usual de summation resultante ab acceleration cardiac.
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Thou, wondrous Harvey, whose Immortal Fame,
By thee instructed, grateful Schools proclaim,
Thou, Albion's Pride, didst first the winding Way,
And circling Life's dark Labyrinth display.
Attentive from the Heart thou didst pursue
The starting Flood, and keep it still in view,
Till thou with Rapture saw'st the Channels bring
The Purple Currents back, and from the Vital Ring

SIR RICHARD BLACKMORE, Creation. A Philosophical Poem Demonstrating the Existence and Providence of a God. In Seven Books. 8vo. London, 1712. [Blackmore, who went from schoolmaster to physician in ordinary to William III ("His pupils grew blockheads and his patients died.") was violently attacked by Pope, Dryden and Swift, but nothing gagged his muse, and the equally intemperate praise lavished on the "Creation" by Dr. Johnson, Addison and Dennis seemed to justify him.]
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