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Auscultation

Introduction - Auscultation was once the premier methodology of cardiac diagnosis, ~~there were~~ ^{several} reasons. It provided the most direct link to the heart and great vessel ^{evidence} of both normal and abnormal function was evaluated by one of the body's most discriminating sensory modalities. For example the ^{presence of} vibrations at the body's surface which produce sound can be readily detected by the human hand but approximately ~~on~~ one million times as much force is required for detection by hand as by the ear. Moreover even untrained ears can detect frequency differences of a few percent and differences in time ~~of~~ comparison of events of a few hundredths of a second.

With advancing technology which allows the direct measurement of heart to be probed by an ultrasonic beam and direct images to be reproduced, & flow to be measured at rates of ~~a ms~~ in times of a millisecond to say nothing of invasive technology which allows measurement and visualization of form and function, it would appear that auscultation is ready to become another

quaint page in the history of medicine.

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~~Technology has~~ The age of auscultation is far from over for the equipment which rivals the stethoscope and a trained observer ^{is large & complex} costs hundreds of or thousands of dollars for each examination. This is in contrast to an instrument that costs ~~only~~ can be carried anywhere in a pocket or more often worn around the neck as a badge of distinction.

A physician who is going to ~~have~~ diagnose or treat patients with a range of ills which include cardiovascular disease simply cannot personally diagnose, treat or follow up treatment of such patients. There may ^{also} be emergency ^{ies} in which auscultation is essential for proper treatment. In short ^{the abilities of a doctor} ^{copying for} a doctor treating such patients is incomplete & basic skills in auscultation.

The only ~~re~~ problem in auscultation is that for the majority it ~~is~~ requires considerable time and effort to learn and some practice to maintain skills. Conversely the same technology which appears to threaten this art is has in fact enhanced its value for we have

learned so much more about the ^{cause &} significance of many auscultatory events. This in turn has allowed us to simplify the process. Technology also ^{now} provides a number of training aids. [3]

The following is an introduction to auscultation ~~takes into~~ uses the advantages provided by current knowledge & technology.

II. Generation and detection of cardiovascular sounds.

Cardiovascular sounds are produced by vibration of the heart various areas of the heart and large vessels. These vibrations ^{from} ~~are~~ produced by disturbance ^{alterations} of blood flow ~~and~~ are carried to the body's surface by various tissues where they produce sound waves which are picked up and carried to the examiner's ears by ^a stethoscope.

All cardiovascular sounds are low in frequency, ranging from the lower limits of human hearing, approximately 15 ^{Hz} ~~Hz~~ to a few hundred ^{Hz}. They consist ^{primarily} of Heart Sounds, Murmurs & bruits, and a few miscellaneous sounds such

as rubs and hums.

Heart Sounds^⑥ are brief discrete events that occur in a fixed time ^{sequence} frames throughout the cycle. They are usually brief and low in frequency.

Murmurs^⑥ occur are longer events that occur in ~~times~~ in sequence but are more complex than H.S. ~~variation~~ ^{varying variation in} in waveforms and frequencies.

~~These~~ These two categories not only differ in characteristics origin as well as in characteristics. Audible events in the heart are usually related to and associated ~~in~~ the four valves of the heart. Sounds Cardiovascular ^{vibrations} sounds from areas around the four valves are most efficiently transmitted to certain areas of the body where they are best heard. ~~These areas~~ ^{as} are shown in Fig. 1.

~~The first step in auscultation is listening~~ ~~In this discussion the~~ Current practice usually describes the areas of auscultation in anatomical terms ^{Fig.} ~~Table 1~~ but the traditional designations are used here for the knowledge they

convey. If an event is heard only in one area it is likely to be associated $\bar{c}$ the valve or structure in that area. ^{However} ~~Conversely~~ sounds from ^{any} other areas may also be heard in ^{an} other areas as well. It is ^{also} good practice to ~~str~~ "inch", or slowly move the stethoscope while listening, to ~~str~~ around an area until the desired sound is best heard.

Normal Heart Sounds ^{IP} All heart sounds are produced by ^{sudden} deceleration, ^{stoppage or slowing,} of a blood stream. This includes the three categories:

- First and Second sounds, S_1 and S_2
- Second and Third sounds, S_3 and S_4 , plus pericardial knock
- Abnormal sounds of valve openings

~~The~~ First and second sounds are doubly important for they have ^{both} intrinsic value and are ^{also} crucial in the timing and identification of other sounds and ~~on~~. ~~The~~ S_1 and the S_1 to S_2 interval defines systole and while it is normally easy to determine this interval at low rates when it is less than half the total cycle this change is reversed $\bar{c}$ increasing rates.

Caution: Auscultation of any area should only be done with initial simultaneous palpation

of the carotid or apical impulse to positively identify S_1 , which occurs just prior to the pulse or impulse and S_2 which follows them. 16

Normal Heart Sounds:

S_1 and S_2 occur when pressure gradients are reversed across ^{inlet & outlet} are associated ^{with} closing of the paired inlet valves, mitral and tricuspid for S_1 , and closing of the aortic and pulmonic for S_2 . Sound producing vibrations are generated. These valves close produced when pressure gradients across the valves are reversed and a backward flow of blood begins. This 'picks up' the valve leaflets which close rapidly, suddenly stopping the initial blood flow. Pressure upon this arrested column of blood cause the elastic valve + its attachments to be displaced and then to recoil ^{producing the vibrations} of blood + tissue we hear as S_1 + S_2 . several cycles of oscillation. Unless the leaflets are heavily calcified actual coaptation of the leaflets is not heard, rather we hear the vibration of blood, valve and especially its attachments.

Normal Heart sounds: One should begin auscultation at the aortic area.

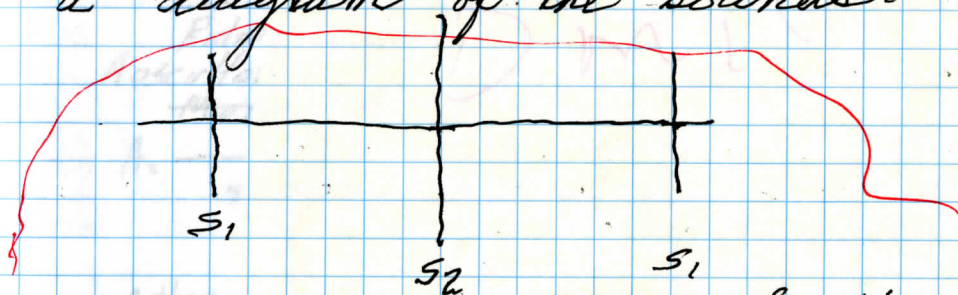
An important aspect The diagrams are the most efficient ^{+ effective} way to document auscultation. ~~They~~

→ begin with a notation of the area A, P, Tr, M; followed

by a horizontal line representing time, beginning at the left. ~~Next~~ All sounds associated $\bar{c}$ values are represented as vertical lines whose height is proportional to intensity or loudness of the sound - these are placed in sequence on the line.

Fig 2 illustrates this process. After $S_{1,2}$ are placed S_3 s and S_4 s, if present are added followed by sounds of abnormal valve openings. Finally, murmurs are added as described in ~~that section~~ ^{section} ~~the section on (pm)~~.

(Left side) Auscultation should begin with the Aortic area. Palpate the pulse and identify the first S_1 . Now compare it to S_2 and draw a diagram of the sounds.



(Opp side) Note that in this location S_1 , closure of the mitral, called M_1 , is heard is not as loud as S_2 or closure of the aortic valve, called A_2 .

Note that S_1 is slightly lower in pitch than S_2 . "Lub-dup" tries to convey this phonetically.

This is not surprising since closing pressures at the mitral are small compared to those of the aortic valve.

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~~Basic Cardiovascular Auscultation - track list~~
~~(L side) Listen to and palpate track 3, a normal pulmonic area.~~
~~(R side) Again the~~

~~Next~~ (L side) Go to track 2, a recording from the pulmonic area of a normal subject, and listen to, palpate and diagram the sounds heard.

Page 12

(R side) Again S_2 is louder than S_1 , but at times ~~the~~ closure of both the aortic, A_2 , and pulmonic valves, P_2 , may be heard as a 'split' sound on inspiration since we are now listening in the pulmonic area where such sounds are selectively transmitted. In ~~patient~~ auscultation the rise and fall of the abdomen should be observed to time this split. Normal splitting which is caused by an increased volume of blood in the (R) ventricle and (later closure ^{of pul. valve}) and decreased volume in the (L) ventricle (earlier closure ^{of aortic valve}) induced by increased neg. pressure in ^{the} thoracic cage on inspiration.

Such splitting may not be heard in many normal patients.

< (L) side > Go to track 3, a recorded ^{ed} ~~ed~~ ¹⁰ from the tricuspid area of a normal subject and listen, palpate and ^{then} diagram the heart sounds.

< (R side) > Relative amplitude of S_1 and S_2 is now reversed since both mitral and tricuspid valves are closer to the stethoscope and their sounds ^{on closing, called} M_1 & T_1 are preferentially heard in this area. While the sound of the mitral closing under the more rapidly rising pressure of the L ventricle is still loudest, ~~but~~ the tricuspid is often heard here as either ^{as} a fused, broadened S_1 , or ~~also~~ as a tightly split S_1 .

< (L) side > Go to track 4, a record of normal sounds from the mitral area, often called apex or apical, and ^{listen} ~~to~~ palpate ^{then} and diagram the heart sounds.

< (R) side > Again the S_1 is louder than S_2 since sounds from the large mitral valve, M_1 , are well conducted by the ventricle to this area.

While there are many variations in the normal heart sounds of normals the

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basic pattern has just been observed and ~~are~~ may be seen in summary when the diagrams are compared. If you found differences between the diagrams you drew & the ones shown, go back to the ~~area~~ area, listen carefully & try to reconcile any differences.

^{Fig 14}
Normal Variants - Two additional ~~son~~ auscultatory events are frequently heard in normals, a ^{third} heart sound, S_3 , and ~~a~~ ^{the} innocent murmur. ~~in the~~ ~~the~~ this normal S_3 is ~~discussed~~ examined under S_3 and S_4 's and the innocent murmur under murmurs.

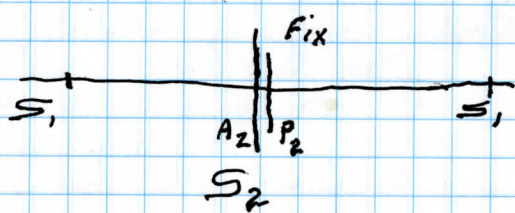
③ Abnormal S_1 and S_2 :

There are many ~~as~~ variations in both amplitude and timing of these sound, produced by a variety of abnormal conditions including 'soft' sounds from decreased ventricular contractility ~~or it~~ frequently associated produced by myocardial ischemia. Timing anomalies ~~can~~ produce fixed and paradoxical splitting of the pulmonic S_2 . Both hemodynamic and electrical ~~patholo~~ problems can ~~prodo~~ cause

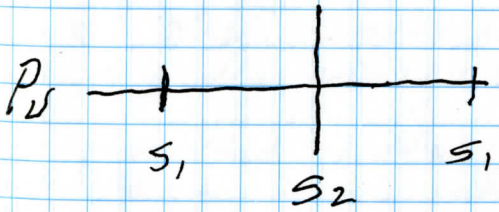
such anomalies. A loud ^{pulmonary} ~~tympanic~~ S_2 can result from hypertension. Any of these ~~there is~~ ^{little} or no flow into the ventricle and diastolic flow into the ventricle and the aortic leaflets are floating. Such anomalous sounds are seldom pathognomonic and their cause must be sought in the associated signs and symptoms of the patient.

Some typical examples are:

(L side) rack number is from the pulmonic area of a myocardial infarct sub pt. Listen, palpate and diagram the sounds.

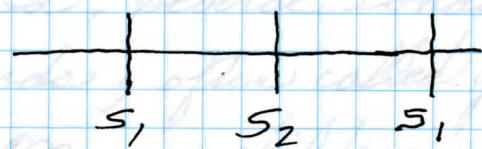
(R side)  S_1 is difficult to hear and the carotid pulse is necessary to locate it $\bar{c}$ assurance. Pulmonic closure is delayed throughout the respiratory cycle by a R.B.B.B.

(L side) Listen to, palpate and plot these sounds from track — recorded from the pulmonic area of a patient $\bar{c}$ pulmonary hypertension.

(R side) Note the loud second sound produced by the forceful closure of a pulmonic valve under abnormally high pulmonary pressure. 

Go to Truck — then
<(L) side> Listen to, palpate & diagram the sounds from the aortic area of a pt. w cardiomyopathy.

<(R) side>



In this case, myocardial ~~ventricular~~ contractility is reduced producing a soft S_2 which almost reverses the usual ratio of S_1 to S_2 loudness.

S₃, S₄ and pericardial knock 14

All of these ^{diastolic} heart sounds ~~are~~ have a common origin, a rapid longitudinal shove or acceleration and brief oscillation of either $\textcircled{D}$ or $\textcircled{L}$ left ventricle from incoming blood. Several conditions can produce such sounds, often called gallops.

Normal S₃: Many children and a number of ~~adults~~ adults, to $\frac{3}{4}$ years old have a slightly hyperdynamic circulation. In most individuals when mitral and tricuspid valves open the rapid inrush of blood is accommodated by a simultaneous volume increase of the ventricle. In some of the individuals the inrush of blood is too great to be totally accommodated and the excess momentum displaces the ventricle. This may be either right or left. All S₃'s follow ^{S₂} opening of the inlet valves in otherwise normal hearts.

Physiologic S₃'s occur when there is a more rapid circulation as in later stages of pregnancy, thyrotoxicosis, etc. and disappear when the ^{pts.} condition returns to normal.

Pathologic S₃'s occur:

- When abnormal ^{volumes} amounts of blood rapidly enter the ventricle as in volume overload. either the ventricle may be otherwise normal or abnormal.

- Ventricular walls are stiff, non-compliant, and cannot rapidly expand as in myocardial ischemia or in hypertrophy. Incoming volume may be abnormally high, normal or even low.
- Ventricle is distended as in volume overload from failure. This also reduces compliance.

S_4 s are usually produced late in diastole when with atrial contraction a rapidly injects a ~~sm~~ normally smaller amount of blood.

It is the conditions are otherwise exactly the same as in production of S_3 's.

However, it is questionable whether S_4 's ever occur in normals.

In summary S_3 's occur early in ^{dia} systole while S_4 s are late in diastole. They are similar phenomena produced by a mismatch of ventricular accommodation and diastolic filling blood flow into the ventricle.

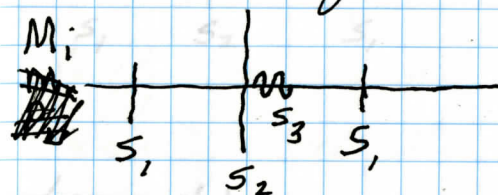
~~Acoustic quality~~ Acoustic characteristics will now be examined:

(1) Trach is from the mitral

area of a normal athlete.
~~Two~~ Diagram the heart bounds after palpation + auscultation.

(R side)

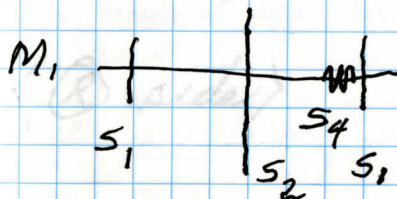
Heart rate is slight increased 84 BPM, and ¹⁶ ~~but~~ $S_{1,2}$ are ~~normal~~ but after a short but ~~amplitudes~~ are reversed, significant interval S_2 is followed by a dull low pitched sound, almost a thud, the S_3 . These ^{two} characteristics aid in separating it from either split S_2 or opening snaps both of which are higher pitched and usually follow S_2 more closely. There may



~~resp. This is from the right ventricle and there may be respiratory variation on the R side.~~

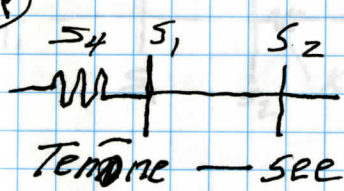
(L Side)

pg 14



Track — is from the mitral area of a patient. Listen to, palpate & diagram the sounds.

(R)



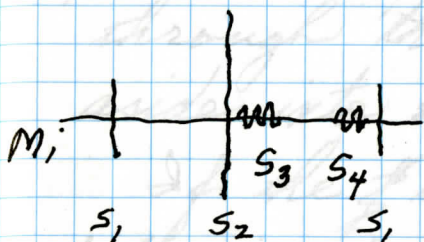
Amplitudes of $S_{1,2}$ are ^{coincidentally} reversed. Late in diastole prior to S_1 a low pitched thudding sound is

heard, an S_4 . There is a popular tendency to think of S_4 as coming just before S_1 , which in fact it does, as atrial preceded ventricular contraction. Horse & buggy doctors called this a "Tennessee" gallop. This sound must be distinguished from ejection sounds which closely follow S_1 & are higher in pitch.

<L side>

Both Listen to, palpate ^{track} and diagram 17
the its sounds from the ? — area of a
patient.
pg 14

<R side>

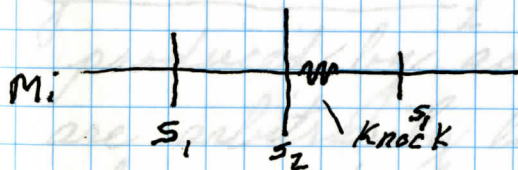


Both S_3 and S_4 are present in
this patient. If heart rate
increases and diastole shortens,
 S_3 & S_4 may merge to produce
a loud summation gallop consisting
of S_1, S_2 & $S_{3,4}$.

<L side>
pg 14

Track 4 is from the mitral area
of a patient $\bar{c}$ pericarditis. Auscultate
and palpate it and diagram the ^{heart} sounds.

<R side>



Loudness of S_1 & S_2 are reversed.
but Note the low pitched sound
following S_2 , somewhat similar
to an S_3 . Mechanism Origin
of the sound is similar
equivalent to that of S_3 but
constriction of the ^{heart} ventricle subtly alters
the acoustics.

Sounds associated with abnormal valve openings.

Normal valves open quietly as blood pressure builds behind the leaflets and blood is driven by this pressure rapidly accelerates to full flow ~~there~~ through the leaflets which are pushed aside into their fully open position.

If the valve is flexible but cannot open fully, is stenotic, then it initially opens quickly & quietly but the restriction brings it to a sudden stop, only partially open. A portion of the accelerating blood stream is suddenly stopped in an action similar to the opening of a parachute. The valve ~~disappears~~ and by the valve, which is now displaced by pressure from the blood, and it, its associated tissue and blood undergo a brief oscillation which we hear as an ejection sound or opening snap. Sounds produced by aortic and pulmonic valves are arbitrarily labelled ejection sounds. Obviously they must closely follow S_1 . Opening sounds of the mitral and tricuspid valves are called opening snaps and closely follow S_2 .

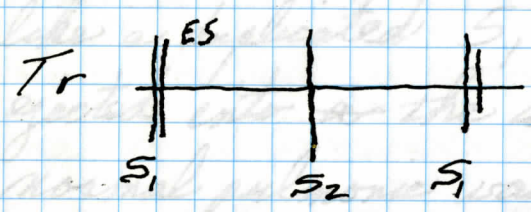
1. A second sound from an entirely different source is also labelled ejection sound. This sound is produced when a ventricle rapidly ejects its blood into a dilated aorta or pulmonary artery usually through a normal valve.

He will now examine the auscultatory characteristics of these sounds. Note: These sounds from stenotic valves are usually followed by its associated murmur.

~~(L)~~ (L) Listen to, palpate and diagram the sounds on track $\frac{25}{2}$, from the $\frac{Tr}{2}$ area of a patient $\bar{c}$ suspected pulmonary stenosis. The sound was louder at the pulmonary area but accompanied by a (m) . This area was chosen for clarity of the sound.

pg 14

(R)



Relative amplitudes of S_1 & S_2 are not normal for this area.

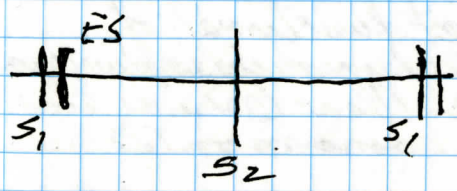
Note that S_1 is closely followed by an almost similar sound, the opening of a stenotic pulmonic valve. This can be distinguished from an S_4 by its higher frequency and closer coupling to S_1 , and of course it follows S_1 .

(L)

pg 15

Track 28 is from the aortic area of a patient $\bar{c}$ aortic stenosis.

Try to ignore the (m) and palpate while listening and palpating and then diagram only the heart sounds $\bar{c}$ the (m) $S_{1,2}$ are normal but note



the sound immediately following S_1 , the ejection sound from $\bar{c}$ partial opening of a stenotic $\bar{c}$ valve.

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S_2 is loud and tympanic secondary to the abnormally high pulmonary pressures which have dilated this main aorta until its a pulse is palpable. What sounds like a duplicated S_1 is the sound from blood ejected into a the dilated pulmonary artery through a normal pulmonic valve. S_2 is closely split & variation

like a duplicated S_1 , is the sound from blood ejected into ~~a~~ the dilated pulmonary artery through a normal pulmonic valve. S_2 is closely split & variation

Murmurs ^{decelerated} (and bruits)

21

In some of the heart sounds are the result of the force of blood ^{on} valves & tissues, ^{all} ^{stream} ^{itself}.
1 murmurs are produced by the blood when it has been accelerated to a ^{high} velocity, ~~too high~~ too high for the situation. When this happens the normally smooth flow breaks into a series of eddies, a miniature whirlpool of diminishing size which produce a spectrum of vibrations whose shape is ^{essentially} the same for all murmurs. The differences we hear is determined by two factors;

First - the intensity, loudness, of a given murmur is related to its velocity of blood.

Secondly - ~~any~~ this ~~deter~~ primarily determines shape of the m

Second - the frequency characteristics are determined by the state and nature of the it passes ~~it~~ which transmits ^{it the vibrations}. A tense ventricle or aorta will include high frequencies which characterizes the produces the blowing characteristics of many murmurs. Conversely the murmurs transmitted by a relaxed ventricle contain only the lower frequencies which characterize produce the low pitched 'growling' characteristic of say a mitral stenosis.

Murmurs may develop whenever blood is accelerated to high velocity, either in normal passages or when ~~or~~ passing through ^{abnormal} constrictions in valves or in vessels. Add- Diagramming Murmurs. 22

Again one of the best ways to document and understand ~~is~~ ^{is} to diagram them. One must begin ~~to~~ ^{the first} diagram of heart sounds for the area under study. start

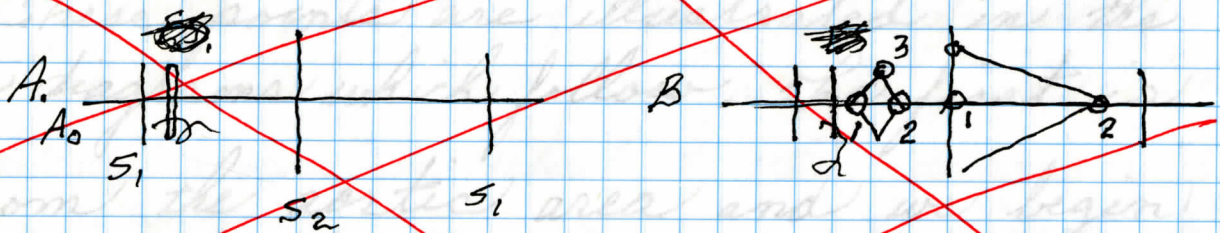
~~1. An introduction to the major murmurs follows. 23~~

~~It begins with a important and often difficult ~~for~~ problem the innocent ~~m~~.~~

~~While the ear is ^{always} drawn to the ~~m~~ or abnormal sound a properly conducted ~~C.V.~~ auscultatory exam of any area begins with ~~absolute~~ ^{certain} unequivocal identification of S_1 followed by S_2 . This establishes systole and diastole. Without this identification listening to ~~a~~ ^{sounds or} ~~m~~ is a waste.~~

~~Next all other sounds should be identified for ^{abnormal} sounds are often the key to identification of a ~~m~~. This should be kept in mind while diagramming sounds and ~~m~~.~~

~~Diagramming of ~~m~~. After all sounds are in place identify the ~~m~~ as systolic or diastolic. Next identify. Now identify the Fig. 3.~~



~~1. If both are present choose one and complete its analysis before starting the other.~~

point in time at which the (m) starts ①; stops ②; and is loudest ③. Now proceed to the

3C



diastolic (m) and repeat the process start ①, stop ② and loudest ③. it is likely that valve is involved

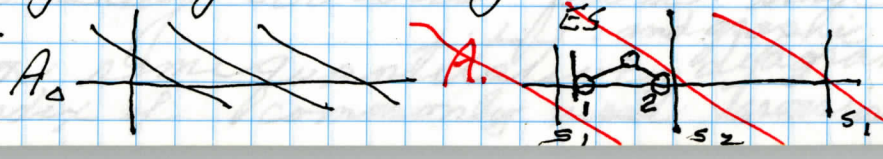
At this point interpretation of the can begin. The murmurs were loudest at the aortic area and

In this example the systolic (m) starts early and stops well before S₂. This indicates the period of maximum flow velocity which climb to a maximum and then decreases. Such a flow pattern is normal consistent with normal flow pattern from a heart at rest.

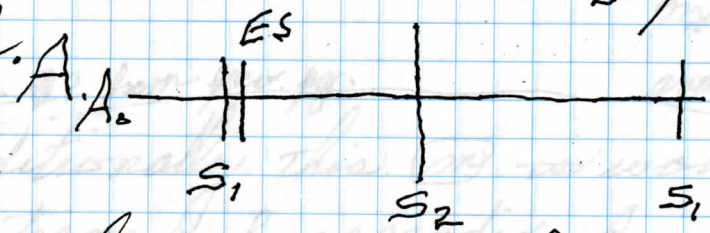
Next consider the pattern of the diastolic (m) ??

Finally, note the character of the (m). Is it high pitched, blowing in character or is it a low pitched coarse grumble. This should be indicated.

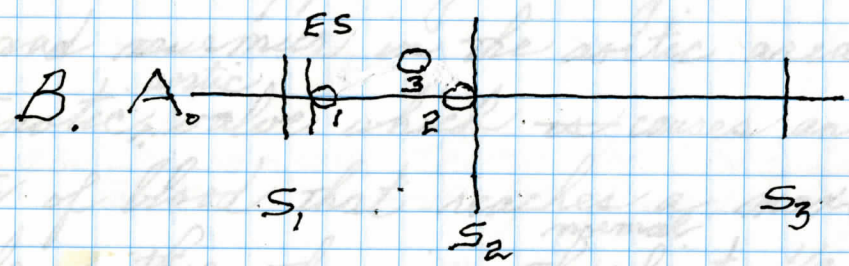
These points are illustrated in the two diagrams which follow. The first is from the aortic area and we begin by completely diagramming all heart sounds as in A.



~~(Q)~~ Track ~~was recorded from the aortic area~~ of an ~~apparently healthy male~~. ~~System after~~ which comes from the aortic area. There are ~~normal~~ S_1 and S_2 plus an ejection sound.



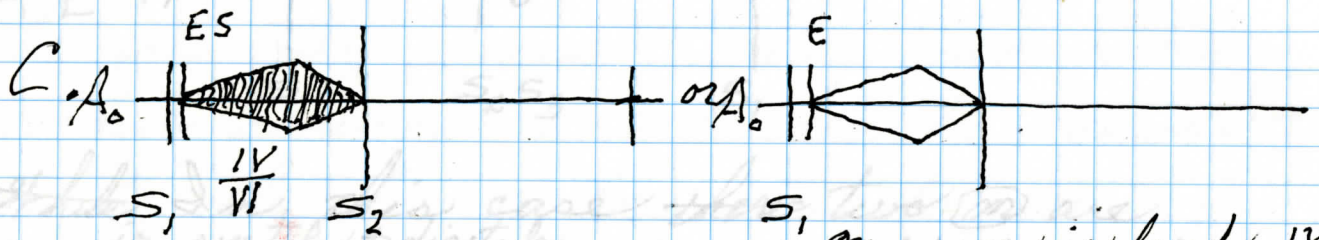
In this case we hear a relatively high pitched murmur which starts at the ejection sound indicated by ① in B. It stops at S_2 as indicated by ② and it is loudest ~~near~~ near the end of the (m), at ③.



The diagram is completed by joining the points, 1, 2, 3, of the (m) and either ~~indica~~ to outline its envelope, C., and either adding words or detail to the diagram to indicate its character and loudness. It is immediately obvious from such a ~~diagram~~ ^{and graphic} semi-quantitative ^{diagram}.
1. In appendix of commonly used terms is attached

the nature of the m -

26



Foot note 2 from prev. pg.

murmur is loud $\frac{14}{VI}$ and medium frequency

Traditionally this m would be described in the terms of appendix A in a few sentences which would then be visualized as well as possible. While this exercise is not primarily concerned with detection and description of sounds and murmurs and not diagnosis is secondary but such a diagram ~~allows~~ ^{conveys} a graphic immediate insight into what is happening; ~~an eye for the~~ ^{the} an ejection sound and murmur in the aortic area indicates a stenotic ^{aortic} valve which causes an ~~increased~~ ^{normal} increased velocity of blood that reaches a maximum late in systole rather than in the first $\frac{1}{3}$ of systole.

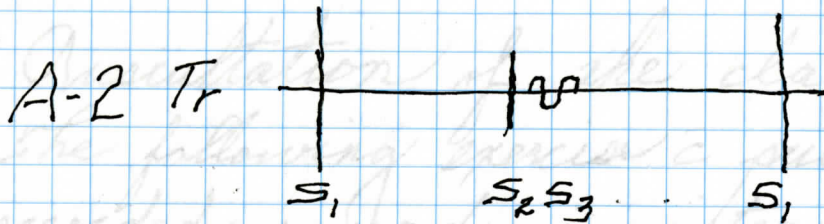
To further illustrate the process of diagrams a more complex set of auscultatory events from the tricuspid area will be diagrammed.

Sounds as shown in A-2 are normal $S_{1,2}$ and an S_3 .

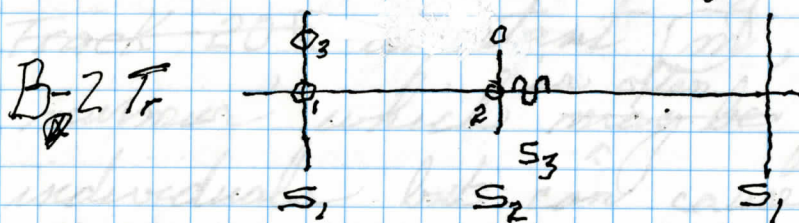
3. The diagrams are usually drawn double sided like a phonocardiogram

3.

2. This is a ~~so~~ entirely consist $\bar{c}$ mod. to severe aortic stenosis.

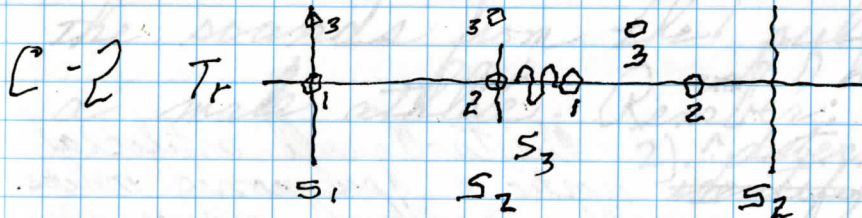


While I'm in this case there two $\odot$ are discovered, ^{in systole + diastole} and again it is critical to determine which is in systole and which is in diastole by palpation. Begin $\bar{c}$ systole; it starts at S_1 and ^① stops at S_2 ^② and is almost equally loud ^③ throughout ^④ Add this information to the diagram B-2.

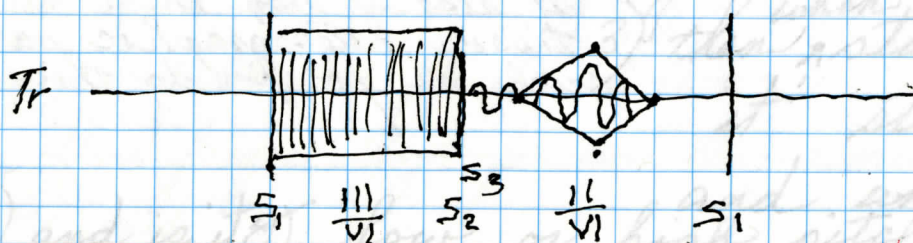


coarse low pitched

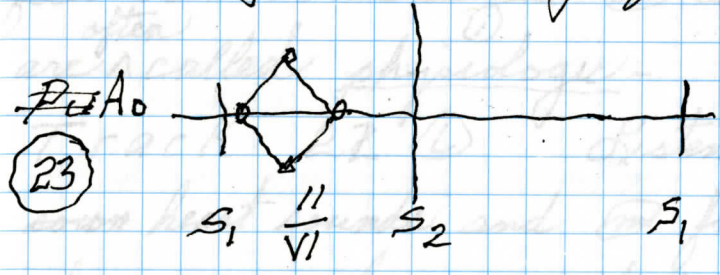
Now do the same thing to the diastolic $\odot$ which starts at the $\bar{c}$ ③, ①, ends before S_2 ②, and



peaks between, the ③. Connect these points, add indications of frequency & intensity - C-3.



Track 20 (right) Only S_1 + S_2 are present and normal. The m is systolic and begins shortly after S_1 and ends before S_2 . It reaches a peak between the two.



Also it is not very loud and has higher frequencies in it.

Left To understand this ^{on track 20} murmur which is often called a flow or ejection m one must understand that $\frac{2}{3}$ of R or L stroke volume is ejected in the first $\frac{1}{3}$ of systole and that ~~not normal~~ m can develop in ~~normal~~ vessel across ~~normal~~ valves and in normal vessels with relatively small increases in flow. In this case we can see that the m ^{is loudest} peaks early in systole indicating that flow is at a maximum - when it should be. ~~There is no other~~

In older people the valve aortic or pulmonic valves may become thickened, "sclerotic", but and produce the above ^{normal flow rates} pattern, but unless the valve obstructs flow ^{this m} is also considered innocent.

One frequently finds One sometimes finds

* Add

~~One~~ Some Oftentimes one finds similar 30
m's when systemic flow is increased ~~and~~
as in pregnancy or thyrotoxicosis. When the increased
flow is corrected the m disappears. ~~and~~ such m's
are ^{often} called physiologic -

Track 22 ① Listen, palpate and diagram
~~soon~~ heart sounds and m from the pulmonic area
of a normal pregnant female in early in her third
trimester.

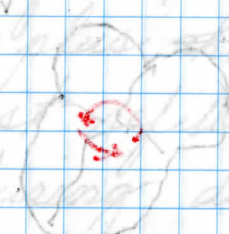
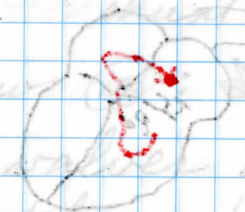
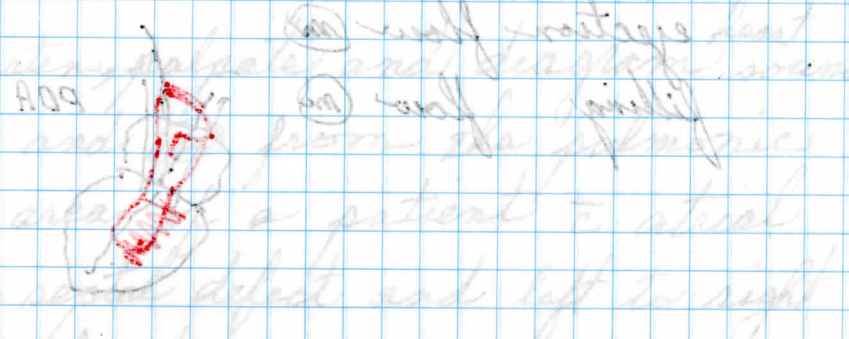
Flow m ^{are fre} associated ~~to~~ associated ~~in~~ ~~shunts~~
shunts and regurgitation: A regurgitant
^{aortic or pulmonic} valve imposes a large volume load on its
ventricle ~~and this is~~ as blood flows from
the circulation ~~to~~ back into ^{in diastole} ventricle. This
extra blood must be ejected during systole
and this frequently produces a characteristic
flow ejection flow m in the aortic and
pulmonic areas. This murmur may be ^{overshadowed} ~~observed~~
by the diastolic regurgitant m but it is
an indicator of severity of regurgitation.
An example of this will be seen on later
when we listen to regurgitation.

Right: Track 21

31

~~S₁ & S₂~~ are normal & normal inspiratory splitting. This murmur is ^{loud} faint but occurs early in systole and does not extend into late systole. It is characteristic of such murmur. This is another "innocent" (m).

~~Flow (m) associated & abnormal flows:~~



If there is a ~~short~~ ^{significant} shunt present an abnormal 32
~~the~~ flow of blood through ~~normal~~ ^{normal} valves of the
heart ~~may~~ can occur. This excess flow
may produce a characteristic ejection m.
For example both atrial and ~~septo~~ ventricular
septal defects will increase flow across the
pulmonic valve.

Track. ~~22~~ ²² (2) Listen, palpate and diagram ^{heart} sounds
and m from the pulmonic
area of a patient c atrial
septal defect and left to right
shunt.

(2) Filling flow m: ~~If excess~~ ^{When} ~~mitral~~
and tricuspid valves are regurgitant, ~~they~~
~~&~~ during systole, and ^{the blood} excess blood they lose
~~must~~ ^{plus} that of that of systemic
flow must refill ~~rough~~ the associated
ventricle during diastole. Such excess flow
causes murmurs ~~semila~~ Diastolic filling.
Two thirds of ^{ventricular} filling normally occurs
during the first third of diastole, so one might

1. This is [!] ~~of~~ ^{the} reversed of regurgitant aortic &
pulmonic valves which leak in diastole &

might expect filling murmurs to start early³³ in ~~sys~~ diastole and be loudest in the first half of diastole. The following is an example of such a filling flow md -

Track 2.3 (2) ~~197~~ Listen, ~~pt~~ palpate and then plot the sounds and murmurs of this mitral area.

~~This murmur was in fact~~

A similar murmur may be heard when there are shunts around a mitral or tricuspid valve which greatly increase flow and filling. In ~~this~~ circumstance an S₃ is frequently present.

Murmurs from Stenotic Valves: These ~~aortic and~~ are the best known and usually easiest - loudest and easiest to diagnose of all murmurs, especially those of aortic + ~~pm~~ mitral stenosis.

~~Aortic and Pulmonic:~~
~~Such lesions~~ The opening (valve area) of stenotic valves usually decrease $\bar{c}$ time. At first

34

there may be little difference between such murmurs and ejection flow murmurs. As the valve orifice decreases in size and blood velocity increases the m starts earlier ^{in systole} & ends later. Also the ~~peak~~ peak flow and ~~murmur~~ ends later & later $\bar{c}$ increasing stenosis and the m peak shifts accordingly.

Track 25 (L)

Listen, palpate and then diagram the heart sounds and m from the area of a patient $\bar{c}$ moderate aortic stenosis.

Track 26 (L)

Listen, palpate and then diagram the heart sounds & m from the area of a patient $\bar{c}$ severe aortic stenosis.

Go to 1st tag P.

Track List. Introduction

Area -	Content	Must Have	Need
1 - Ao	Normal, Normal rate $S_1 < S_2$	✓	
2 - Ao	Normal, Tachycardia $S_1 < S_2$		✓
3 - PU	Normal, Normal S_2 split $S_1 < S_2$	✓	
4 - PU	" " $\bar{S}$ split $S_1 < S_2$		✓
5 - Tr	" " , $M_1 + T_{E1}$ $S_1 > S_2$	✓	
6 - Tr	" " , $M_1 \& T_1$ fused $S_1 > S_2$		✓
7 - Mi	" " $S_1 > S_2$	✓	

It would be desirable to have +, 3, 5 & one each of 4 above areas from same subject.
(Normal Variants)

8 - Mi	Normal $\bar{c}$ S_3	✓
9 - PU	↓ $\bar{c}$ + innocent (m)	✓

(S_3 & S_4)

14 - Mi	'Normal' $S_1, S_2 + S_4$	✓
15 - Mi	" " " " + S_3 & S_4	✓
16 - Mi or PU	" " + Pericardial Knock	✓
17 - PU	S_3 or S_4 $\bar{c}$ respir. var	✓

Abnormal Opening Valves

18 - Ao	S_1, S_2 plus ejection sound	✓
19 - PU	S_1, S_2 ↓ ↓ ↓	✓
20 - Mi	↓ ↓ ↓ O/S	✓
21 - Ao	S_1, S_2 plus ej. snd, From diastole	✓
22 - PU	↓ ↓ ↓ ↓ ↓ ↓	✓

Except 9 all of above should be $\bar{c}$ (m)

Track List Normal; cont'd.

Heart Sounds, Cont'd.

23
+9 - Mi - Mid sys. Click.

Must
Have
✓

Need
✓

31

Murmurs

20 ⁴	A ₀ - AS, mild		✓
21 ⁵	A ₀ - AS, severe c ej. snd.	✓	
22 ⁶	A ₀ - ↓ ↓ S ↓		✓
23 ⁷	A ₀ - AR, mild		
24 ⁸	A ₀ - AR, mod to severe c ej. (m)	✓	
25 ⁹	Tr - TR, ↓ ↓ ↓ ↓ ↓ ↓	✓	
26 ³⁰	Mi - MR c dias. flow (m)		
26 ^{30A}	Mi - MR, mild		✓
27 ³¹	Mi - MS + OS	✓	
28 ³²	Mi - MR + MS		
28 ^{32A}	" - " + " c tachycard.		✓
29 ³³	Tr - VSD	✓	
30 ³⁴	PU - PDA	✓	
31 ³⁵	Friction Rub	✓	
32 ³⁶	Râles -		

Analogous murmurs may be heard in pulmonary stenosis.

Track 27 (L)

Listen, palpate and then diagram the sounds & (m) from the pulmonic area of this patient.

Mitral and tricuspid Stenosis: There are several obvious differences in these murmurs since most of the filling is normally passive, filling pressures are low & the murmurs are diastolic. Another major difference is that the relaxed ventricle filters most of the higher frequencies and only low pitched rumbling sounds of these (m) are heard.

Track 28 (L)

Listen palpate and then diagram the sounds & (m) from the mitral area of this patient who had rheumatic fever.

1. This lesion is now uncommon

Regurgitant (m) including congenital shunts

When valves are incompetent and there is significant retrograde flow, the velocities are high since the orifice size must be small for the patients to survive. Also, except in the case of atrial septal defect, there is a large pressure gradient across the orifice or defect and the murmurs are much less variable than flow

(m). Archetype of regurg ^{Mitral, Tricuspid}
~~Aortic & Pulmonic regurgitation.~~ ^{small} and ^{small} Ventricular
~~Septal Defects~~ produce the prototype of regurgitant (m) for the large pressure gradients ~~for~~ produced by ventricular pressures produce high velocities which persist over most of systole producing a murmur of more or less constant amplitude.

Track 30. Listen, palpate and then diagram heart sounds and (m) from the tricuspid area of this patient a
<VSD> a long standing (m)

Track 31. Listen, palpate and then diagram heart sounds and (m) from the mitral area. After examining First examine the systolic and then the diastolic (m)

Aortic and pulmonic regurgitant (m) are typically decrescendo. Unlike the pulmonary and vena cava ^{carry} which ^{could} absorb the volume of a ventricle backflow from regurgitant of volume from tricuspid, mitral or ventricular septal regurgitation with raising increasing pressure. However when the regurgitant volume from a mitral/tricuspid valve rapidly increases the pressure in the associated ventricle and regurgitant blood velocity is decreased. This reduces intensity of the (m) through out diastole and may stop the (m) well before the end of diastole, S₁.

Track 32 - (8) Listen to, palpate and then diagram the heart sounds and (m) from the aortic area of a patient. Listen first to After the sounds listen to the ^{diastolic} ~~systolic~~ murmur, then examine the ^{one in} ~~systolic~~ ^{one}.

Remember that ^{regurgitant volume of} the ^{leaps} blood which ^{leaps} into a ventricle in systole must be ejected ⁱⁿ the forward ^{system} volume during systole. This may produce the classic flow (m).

Temp Urine

~~There is one regurgitant lesion in which blood flows continuously in both~~

Mitral valve prolapse produces a variable ~~(m)~~ but the typical murmur begins when one or more cusps of the mitral valve fail and open after S_1 under ventricular pressure. This may occur almost anywhere in systole and produce one or more "midsystolic clicks" which are followed by a murmur which continues to end of systole.

There is at least one regurgitant lesion in which blood flows continuously since there is always a pressure difference between the aorta and pulmonary artery. This murmur has regular variations & produced by ~~chan~~ the cyclic changes in B.P.

Bruits: can occur in ^{any} vessels whenever flow ~~exceeds~~ reaches a velocity ^{increased} too to cause turbulence. ~~Also~~ normal or reduced flows may occur when reach turbulent velocity in the face of an obstruction such as atheromatous plaques -

Tr 5 - This is a Right normal tachycardia rate, $\sim 130 \text{ BPM}$ for a normal infant but rates this high are frequently encountered in cardiovascular disease greatly increasing the difficulty of assessing heart sounds and m. S_2 is considerably larger than S_1 . Heart rate

③ more variable
④ are longer in duration, and more complex in structure. ~~For~~ ⁱⁿ than heart sounds. An important aspect of ④ is their ^{variation of} shape, i.e. their intensity $\propto$ time.

A few facts should be kept in mind when examining a mm. Across the population, 9 of 10 mm are benign, are not associated $\bar{c}$ disease. Murmurs in diastole are abnormal, period.

1. Some are continuous beating through systole & diastole while many will occupy systole or diastole.

Murmurs

Increased
Excess Forward Flow
Reverse Flow
Stenotic Restricted "

Pg 17 x Such excess velocity can be produced by three conditions:

- Excess Increased blood flow {local, systemic, transient}
- Restricted ^(stenotic) blood flow
- Reversed or regurgitant blood flow

So called ejection and flow occur whenever blood flow exceeds the turbulence ^{produces} requires a velocity greater than the enough to ^{generate} produce turbulence across an unobstructed valve or vessel. ~~There~~ This may occur in otherwise normal hearts in individuals with hyperdynamic circulation, or increased. Since peak blood flow occurs and velocity occurs in the first $\frac{1}{3}$ of systole or from increased systemic or intracardiac flow. Increased intracardiac flow may be from shunts or regurgitation and may also cause during systolic ejection and rapid phases of diastolic filling. The above may occur on either side of the heart.

To understand ejection and flow it must

be understood, and remembered, that two thirds of the ventricular volumes are normally ejected in the first third of systole, and two thirds of ventricular filling occurs in the first third of diastole. For this reason ejection flow @ occur early to mid systole and diastole.

Stenotic @ occur when a normal or decreased ~~b~~ blood is accelerated to turbulent velocities to maintain normal or decreased flow. Such stenosis may occur across in the aortic or pulmonic outlet valves or the mitral or tricuspid inlet valves. Since considerable pressure may develop to maintain flow such @ may be loud. Also they typically start early and end late.

Bruits may be considered @ in peripheral vessels. They can occur in normal vessels $\bar{c}$ increased flow or in stenotic vessels $\bar{c}$ normal or reduced flow.

Regurgitant @ and @ occur when blood is forced backward through relatively small orifices from regions of high to much lower pressure. They have This may occur in systole or diastole. Such @ have relatively simple but unique characteristics.

Temp Urine
~~On~~ Sounds for Practice (section) then
Track 38 Listen to, palpate and diagram
the C.V. sounds from the mitral area of an
asymptomatic male pilot in for an annual Px.

Put following on opp. pg -

Track 38 - A tachycardia of ≈ 103 BPM is present
and S_1 is louder than S_2 . A ^{third valvular} sound ~~some~~ somewhat
similar to S_2 precedes it later in systole. It might
be confused $\bar{c}$ an S_3 but (compare $\bar{c}$ Track 8) but
the rhythm is different and S_3 s are somewhat
longer, and lower in pitch. ^{best heard $\bar{c}$ the diaphragm} This is a late systolic $\bar{c}$.

There is no associated (m) which is consistent
 $\bar{c}$ little or no regurgitation and lack of Sx -
S.J.

Track 39 - Listen to, palpate and ^{then} diagram the
CV sounds from the aortic area of this 45
Y.O. male $\bar{c}$ history of a heart & murmur.

Track 39 - ^{opp pg}

While S_1 seems normal & a loud banging
or knocking S_2 dominates all other sounds. It is followe
ed by a decrescendo (m) which begins $\bar{c}$ S_2 and

track 39 contd.
continues to S_1 . Such a murmur is consistent
c a moderately incompetent aortic valve which does
not overflow the ~~ventricle~~^{ventricle} and stop backflow ~~and~~ $\textcircled{m}$.
regurgitant flow and $\textcircled{m}$

Track 12. S_2 is louder than S_1 , and also sharp & higher in pitch than normal. Closely following S_1 is a second similar sound, the ejection sound from the flexible but initial ejection of blood into a pulmonary artery dilated by long standing hypertension until its pulse is now palpable - this is a confusing sound that usually must be distinguished from valvular ejection sounds on the basis of other findings.

Jan 2003

16th

Medical -

Prob. Frequency at night - ? f of coffee ?

~10 Jan up 9 1/2 hr. ~~No probs~~ Becomes painful in bladder?, urethral areas sharp & const. $\bar{s}$ red -
No prob. $\bar{E}$ control, starting stream - est. 100-200 ml.
Incomplete emptying on initial void - another 25-50 ml
on second effort $\bar{p}$ 1-2 mins. Clear, colorless -
Last void ~ 6 AM smells & may be turbid but is colorless.

No coffee tea or etoh today - will log for 10 days

16 Jan No Coffee, Tea, ETOH

2155 - void
1245 - 250 ml, 325 ml clv, yellow
0233 525 " 550 "
0544 820 " 925 " 950, 1000

Discomfort Pressure
in penile urethra

7:49 ≈ 7.5 h } 1,000.0
78
220
156
640

17 Jan Void 2330?
0013 200 ml
109 117 525 "
248 810 "
349 275 + 1085
618 550 1360

291
1,8) 5250
36
166
162
30 6.8) 1360.0
272
200.
136

✓ Coffee + Chicory - Large tsp. (~1000)

18 917 Void m
1205 (15) 225 clv. Yellow +
1623 325 " +
2131 530 " +

log 027 350
207 725
435 950

440x 200 722 250 1 cup Coffee + Glass H₂O + Glass Juice
1013 390 2 cups H₂O
1333 550
1730 650

2123 215 265
2355 550 1100
0303 780 1400
0732 1000 1650

20th

202 140 3+
527 320 2+
2143 475 3+ conc

+ 866
+ 0:39 + 0:45

17 Jan - 0200 1 Ft stdg. (R) 1:16 0258 (R) 0:45 (R) 0:41 (R) 0:18

10:21 (R) 1:46 (fatigue)

1400 ~ 5:30 - 7+ / min

1707 (R) 1:15 less waver (R) 0:47

21 047 295 760
0323 528 1000
0546 800 1275

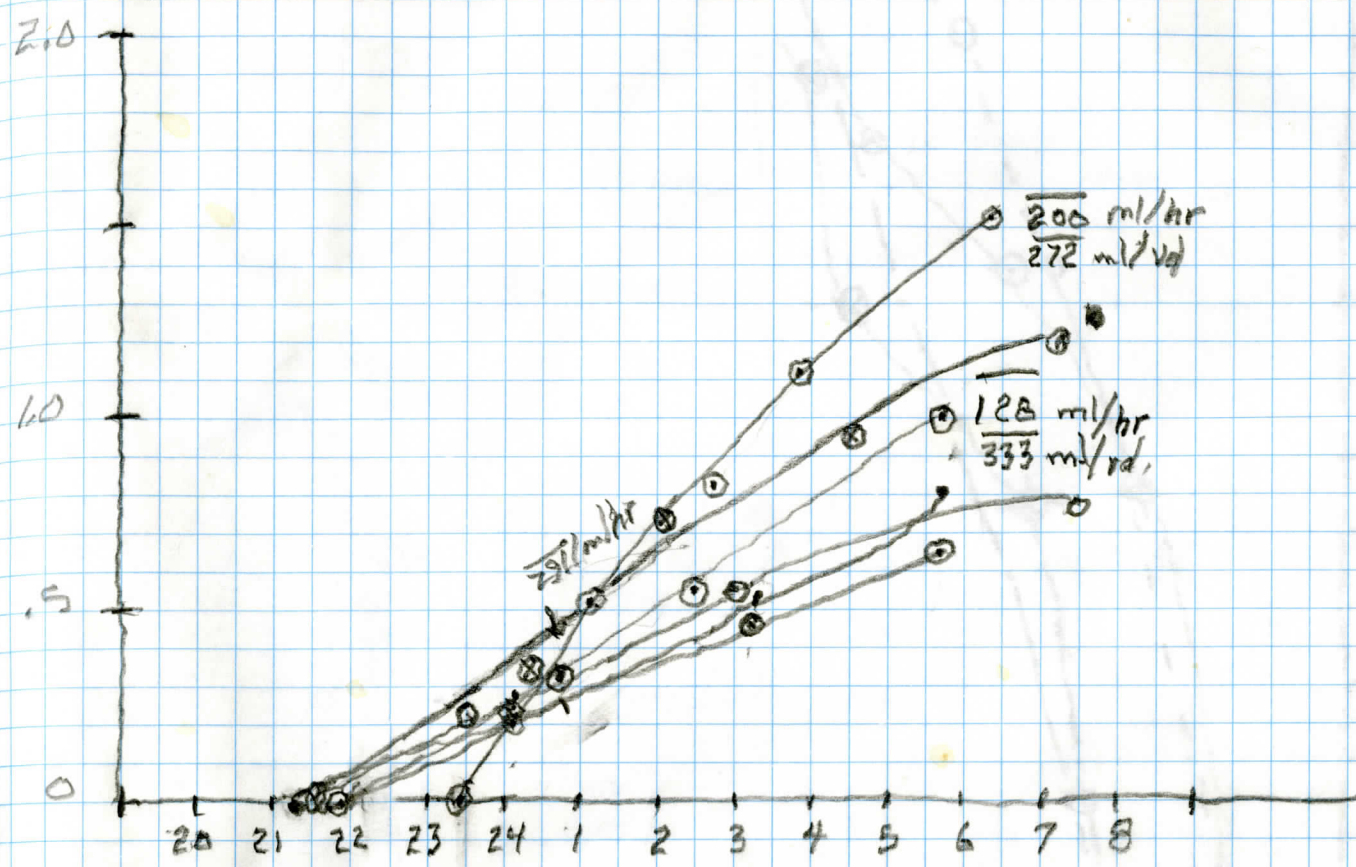
1 cup 1 1/2 tsp Fr Roast + urg. + 2 color
urg. 1+

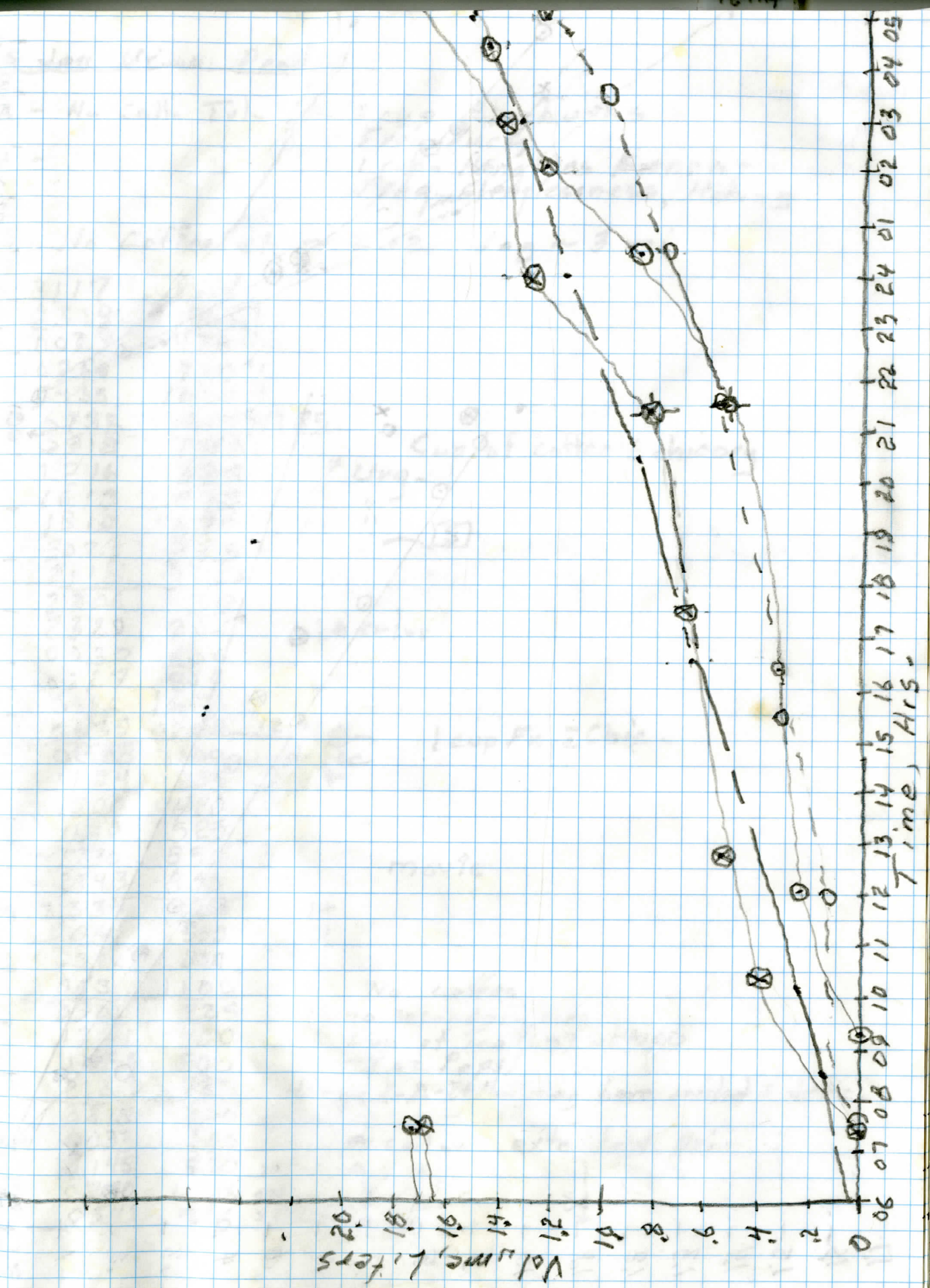
0838 150
1006 250
1613 670
1956 750
2056 880

22 0030 275 1155 1+
0316 470 27 urg → 535-650

1 cup Fr - Chicory
+ Sweet Cond.

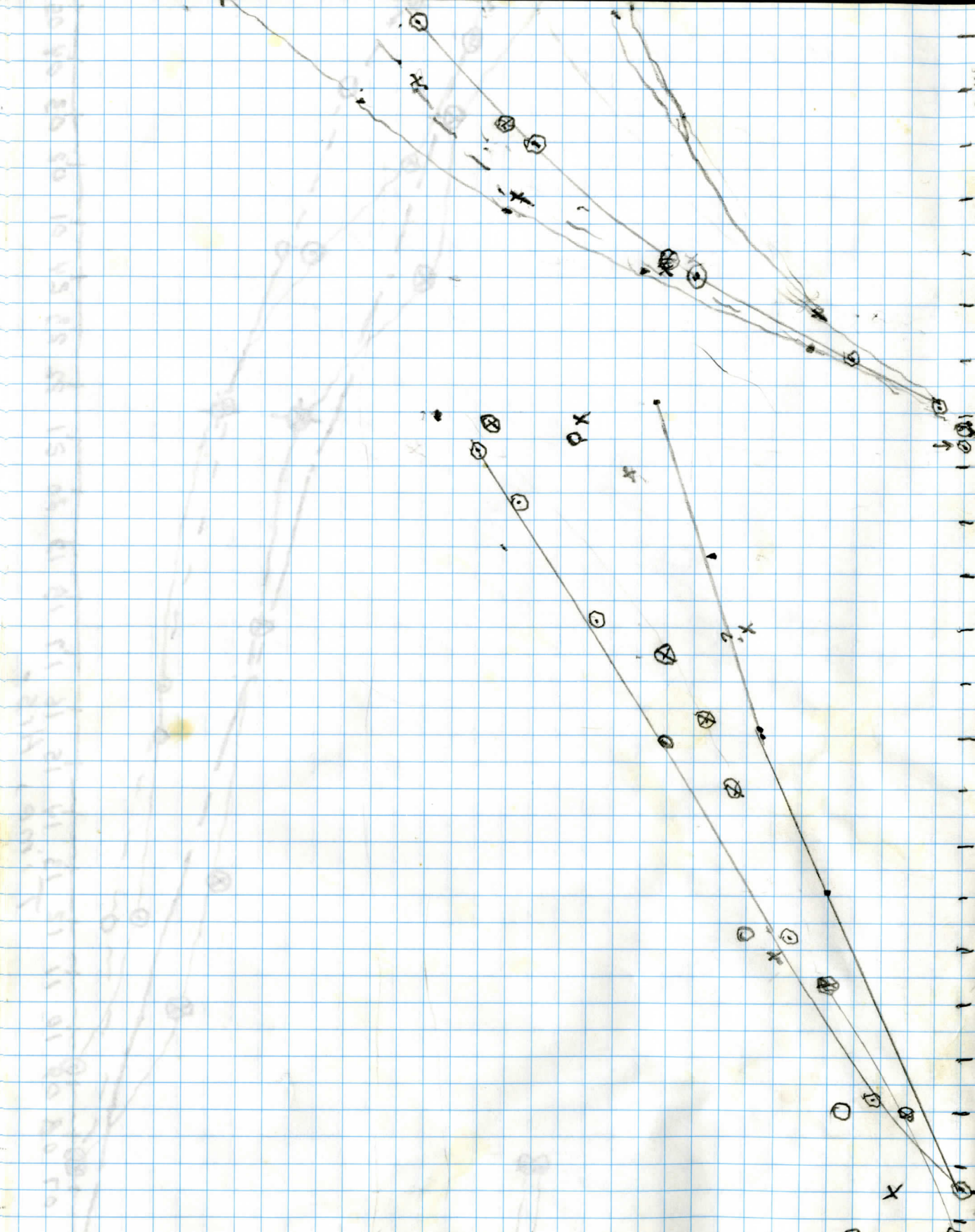
Plot of vols. on P.





17-
 16-
 15-
 14-
 13-
 12-
 11-X
 10-
 9-
 8-
 7-
 6-
 5-
 4-
 3-
 2-
 1-

0600 08 10 1200 1400 1600 1800 2000 2200 2400 2600 2800 3000 3200 3400 3600 3800 4000 4200 4400 4600 4800 5000 5200 5400 5600 5800 6000 6200 6400 6600 6800 7000 7200 7400 7600 7800 8000 8200 8400 8600 8800 9000 9200 9400 9600 9800 10000



25 Jan Urine Prod.

23 - No Coll. Tv)-

1 cup Starbucks

Frequency

24 -

1 cup - Mang Mes French -

Freq. Sleeplessness, Itching

25 No Coffee et ~ 2:02 Jog ~ 3 mi

2117 Void

→ 2319 190 ml

0029 425 "

26 0206 725 "

0435 1025 "

0738 260 → 1285

0918 155

1216 320

1613 475

1810 675

2023 825

2118 900

2206 50

→ 2329 260

0030 500

27 0227 800

0516 1610

0639 1200 1210 0

0838 100 defec

1124 250 2+

1500 420

1610 525

~1730 550

2143 830

28 2332 380 1+

0051 550 1+

0326 850

0630 1100

0900 225

1221 400

1553 500

2127 725

2318 290

29 0033 600

0145 850 2+

0350 1125 275

0541 1375 525

Cup of coffee & chicory

Urg-

"

"

~[S]

1+ color

1 cup Fr & Chic.

movie

No coffee

1+, no secondary urge

Cup of Tea (LS) at 1000

~4 oz Pepsi

Jogged - Δ - 2+ lb - may have voided & defec.

a color extended. Urin.

0 "

0 "

0 "



29 Urine Production

9th 0736 150 ml 1+ urg.
 1150 350 " 4? last void
 1757 50 " 400
 2051 275 " 625 3+ urg
 2151 305 " 655
 2349 275 1+
 30th 0123 575 1+
 0302 825 780

0410 1015 125

0742 1320 430

1310 250

1612 375

1939 460

2215 570

0153 225

0330 525

0638 800

0902 1370

31 1039 190

1204 325

1420 490

1649 715

1939 860

2155 980

1st 0012 350

0015 475

0142 775

0427 1135

0600 1375

1122 260

1530 440

1803 590

2204 725

0029 300

2nd 0208 600

0313 870

0547 1170

0729 1370

0913 200

1330 400

1510 500

1934 655

2218 730

0042 300

0244 535

0502 710

B- Gl. Juice, 1/2 grfruit bowl cereal + lg. banana
 1/2 cup coff + Chk.
 " " " L- Rd. beans/rice - soda brd.
 " " " + Arab. marmalade
 Log 3.1 A - 2.5#

concentrated

1+ painful urg.

1 cup 2 tbsp. Fr. Coffee 241; 3 H₂O 141; juice
 Urg - 2+ 1 cup H₂O

Glass H₂O

" " + Wine

delay

delay.

Urg

Urg

Urg.

est. (void in restr.)

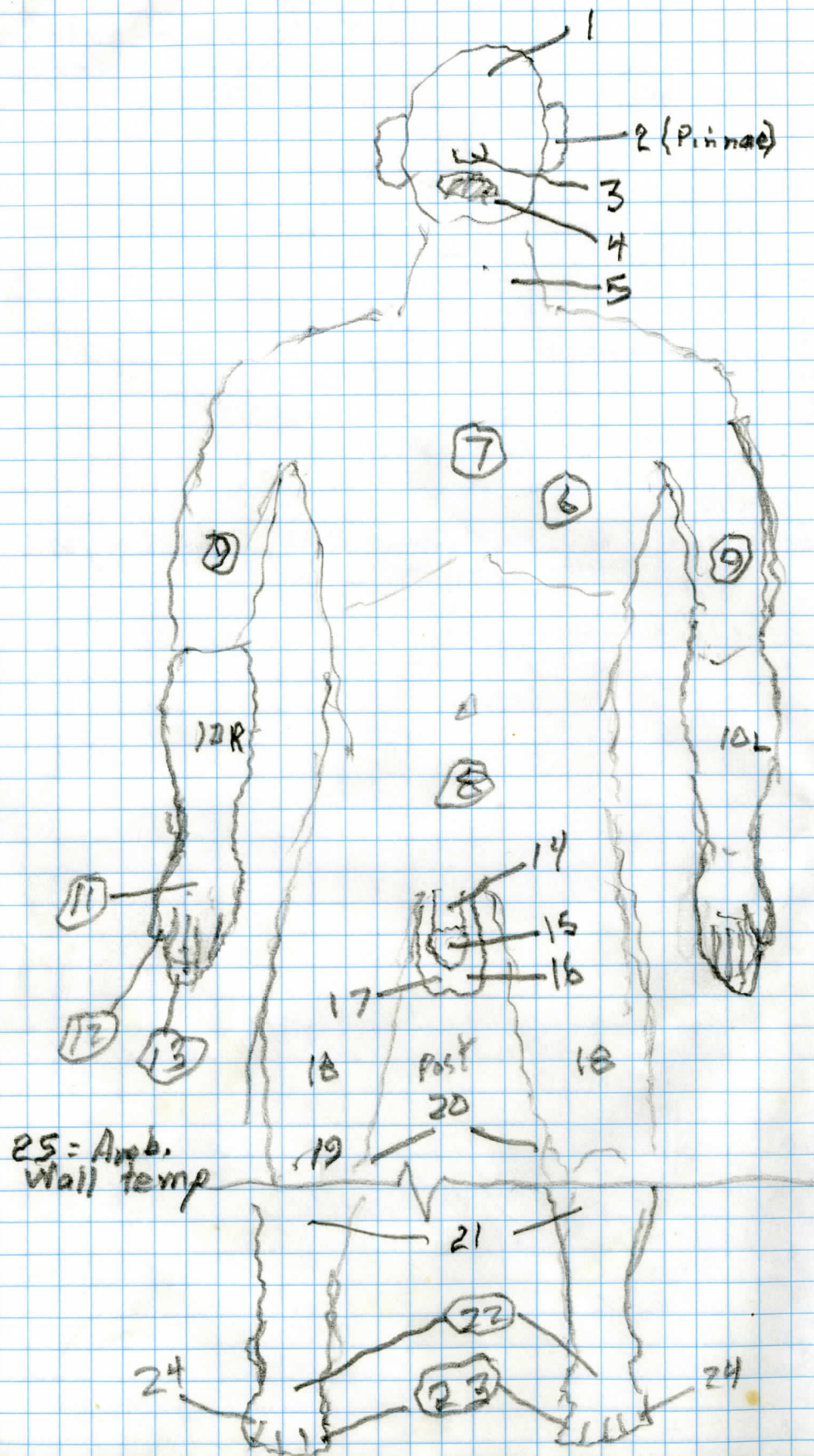
defec. 1 tbsp. Ch./Coffee

Log 3+ mi

2+ color

? cloudy

872B 900



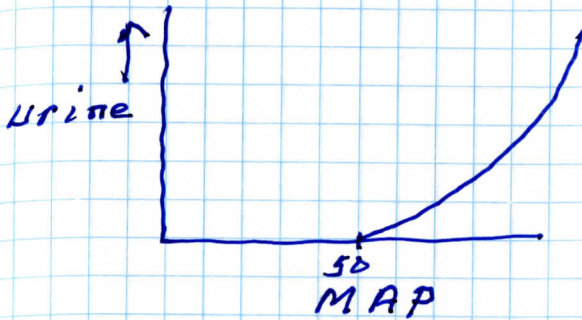
Surface Temp. Meas. & IR Radiometer - Raytek Mini Temp.

1	90		91
2	84/1		93/3
3	81		90
4	98	97	96
5	94		95
6	-		94
7	-		94
8	94	91	93
9	93/4		93/9
10	93/3		94/9
11	83/2		92/9
12	71/3	92	91/8
13	75/2	91	81/8
14	94	95	91
15	91	94	92
16	93	94	94
17	93		95
18	91/8	91	91/9
19	94/1	91	91/9
20	93/3	91	93/9
21	90/1		92/9
22	-		89/9
23	-		85/8
24	-		84/8
25	72	-	72

26 Jan 1220 00T44

C.V. Controls -

Basic - (Earliest) - Fluid Pressure Curve



Renin-Angiotensin

Constrictors - Epi-Nor Epi
Angiotensin
Vasopressin

Dilators Brady Kinin

Local Control -

Time frame -

Role of drugs w

Vase Diuretics

ACE inhibitors

β Blockers

Ca^{++} blockers