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Observations on a rapidly fatal case of pericarditis with large effusion, with treatment suggested to prevent similar results.

The subject of the case was a married woman, age 25. The first acquaintance with the patient was 10 months previously, during confinement, which from a malposition of the child being at the back of the pelvis required the use of the long forceps. This was her first child. From this she made a good recovery. This of course has nothing to do with the pericarditis, but I
Simply mention it as the only occurrence in the history, the subject being previously healthy, it was so from that time to the time of her present attack.

June 13, 1876. I was called to the patient at this date & found that she had complained of slight joint affection for two days. The rheumatic affection itself was a very ordinary attack, there is therefore nothing about it. There were no other cardiac symptoms, but slight pain in the region of the left breast, till the 21st, the joint affection gradually abating for the two pre-
-vious days. This (21st) the first day of the change in the symptoms there was a manifest change in the patient's condition. There was evident distress, increased thirst, lips parched, distressed breathing (not like an ordinary dyspnea). Urgent requests for more air, the eye brighter than natural. Over the base of heart the double friction sound was heard, faintly, for the distress of the patient prevented prolonged examination; the percussion dulness was extensive although no exact measurement could be taken on account of the annoyance it caused the sufferer.
During the night all the symptoms became aggravated. The next morning, 22nd, breathing more distressed, eye brighter, Facifications considerably, orthopnea constant, continual restlessness. This might not rest, but the general distress & that of the breathing, increasing. Slight lividity of the face.

23rd. Patient sitting in bed, head drooping forward, neck flexed acutely, lividity more marked, drowsy. On being spoken to, she looked up, appearing very distressed & anxious. Seemed to have great difficulty in swallowing the
Saliva, which was attempted, to wet the throat. Lips very parched. The eyes were now dull, but on being questioned they became brighter, when she stared wildly around. Face of a fainter livid hue than life. Heart was now too rapid — 130 per minute — to distinguish more than its normal sounds which were indistinct, if being rendered more so by the continually recurring irregularity of the heart.

The distress was now extreme, but not so continuous as on the previous day, being intermitted by intervals of calmer. This of the growing lividity of the face...
Hips indicated that the end was near. Pulse like the heart very rapid, irregular & becoming weaker. The condition of the cerebral circulation being indicated by the mental distress, faintness, breathing wandering & delirium were interrupted by occasional lucid intervals which became shorter less frequent as the case advanced.

Evening visit,—signs & symptoms exaggerated.

4th Morning. The night had been worse than any previous. She having roused considerably from the previous day's semi-comatose.
Condition. Her screaming was so loud as to be heard for some distance in the neighborhood. During the night she refused to take medicine or anything else. On being spoken to she seemed more sensible than on the previous day, but on account of the distress expressiveness of the breathing it was somewhat difficult to understand her interrupted articulation. Heart now very irregular. Pulse about 140–150 being so palpable irregular that the exact number was uncertain when properly roused, the wildness of the gaze was, if
More extreme than yesterday, & the condition was one of extreme distress to look on.

The bodily weakness now increasing, the patient had to be propped up, feared to fall into what was known the pronounced nip; favoured.

This state of things continuing, the coma deepening, with occasional cries & jactitations, the patient expired at 1 P.M.

This is shortly the history of this case so far as it concerns our present purpose, namely, its cardiac origin.

What is principally to be remarked about the case, is the rapidity
Probability could not have been produced on account of the troublesome vomiting of the second day. (I mean of course the second day of the cardiac symptoms).

The only thing I regret in the treatment of the case, is not attempting to drain off the fluid with trochar and cannula. What deterred me from this was the knowledge that patients are said to have died in the physician's hands during that operation. An accident which would be dreadful in its occurrence. However, the after regret of not not having performed paracentesis, led me to think of
a modification of that operation which is theoretically free from
the danger of the operation as at present performed, for
I have not, since this case occurred, had an opportunity
of practising it.

Now of some of the phenomena in the case, of the causes of
the fatal result.

The early occurrence of the rapid increase of the distress of breathing,
the large area of dulness on percussion, the rapidity, brief-
-ularity & transtentous action of the heart all indicated the
abrupt interference of the peri-
-cardial effusion with both
Circulation & respiration.
The irregularity was characterized by one large powerful contraction of the heart beating very strongly against the chest wall, followed by contractions growing less less powerful and more rapid, till the 6th or 7th was reached; then came a long interval which ended, it was followed by the large powerful contraction with its attendant 6 or 7 smaller beats. This condition continued till the pulse got up to about 130, it became more feeble, when neither the long interval, large beat, nor its followers could be so well made out, but there was perceptible irregularity of both heart and pulse.
the resistance being less than the force prepared, the ventricle as I have said contracts violently (this is the action which I have previously called the large thorofall contraction). There is no resistance to the ventricular wall becoming tense on the small quantity of fluid in it, now it effects this onward. This shock or surprise is very quickly followed by the other very more rapid, less powerful contractions, which are quicker and less forceful effectual from two causes. First, the prior shock by the less quantity of blood in the ventricle is explained as there is really no true force
to refill the venticiles. Examples of similar fluids of surprise and shock are seen in manual labor 
machining; as in working an ordinary pump. When one is 
depressing, raising the handle 
rigorously, if by some accident 
a leak occurs in the chamber 
or should one of the valves 
not act properly, we at this 
time working with force 
sufficient to raise the quantity 
of water we had previously 
not accustomed to, this takes 
prepare instead the usual 
amount of force. Meeting with 
very little resistance the handle 
goes down with a bang, and we 
accordingly experience a surprise 
receive a shock proportioned
to the amount of force expended. This corresponds to the large and violent contraction of the ventricle; but we know that something is wrong about repeat the ex- experiment until we acquire into treatise matter. The heart can't do so, but more with effort; actual Contraction follow this shock until the heart as it were gets exhausted; then follows the long pause, to be again followed by the same shock + rapid Con traumatic. We have perhaps a nearer resemblance to the case of the heart in a piece of machinery. Take a steam engine working up to the required power, & driving up to the required mill.
When the mill is equally fed with the spread-out sheets of grain, the engine works quite steadily, but when from any cause the feeding flags, immediately the engine acts faster. In fact, as there is less to do, in fact, as the mechanism becomes lighter; then again it is properly fed and the whole goes on steadily until the cause causes trouble about irregularity in its action. This is not, however, the only irregularity of the heart which may be explained in the same way. Mitral regurgitation, whether that occurs from leakage through a contracted or dilated orifice, gives rise to irregularity which admits of the same explanation. Here
Unlike the half-filled ventricle occasioned by the presence of the pericardial fluid, we have a ventricle quite full in mitral dilatation or nearly so in stenosis unless the contraction of that orifice be very great. We have here plenty of blood to be propelled but the mechanism for its being expelled from the ventricle in the onward direction only is disordered. The ventricle contracts on its contacts to fill itself out of its cavity too easily, thus receiving a surprise shock as in the case of the presence of fluid in the pericardium, the insufficiently expelled ventricle in consequence.
In this case, the heart of the blood, proportioned to the venous picture, flowing backwards into the auricle, while the remaining portion is propelled forwards, but not until the pressure in the auricle (for the valvular resistance is gone or nearly so) resists the further backward flow of blood equals or rather exceeds the pressure in the aorta to be overcome in the forward direction. The pressure in the aorta in this case being below the normal. In consequence of this leakage backward through the mitral valve, in this way the auricle emptied.
itself. The next few contractions form the irregularity, the beats being similar to our case of irregularity from pericarditis with effusion, but not so marked. The state of this irregularity being regulated by the condition, a small amount of the valvular lesion or the work of excitement to which the heart is subjected, to at the time of observation.

To return to our proper subject. Now of the further interference which the pericardial effusion presents to the proper cardiac action. The increasing quantity of fluid aggravates the interference to the ventricular action dilatation. When the heart is surrounded by the fluid, its action is hindered not
only by the quantity of 24 fluid itself, but when that amounts to a large quantity we have superadded, stretching of the pericardial sac, that will occur without the sac being full at the top or apex of the bag, for mere weight of fluid will press out the bag laterally and stretch the membrane. Now what is the effect of this on the Contaminid heart? Suppose we start with the heart at its fullest dilatation that is permitted in presence of this mass of pericardial fluid. The sac is stretched to the utmost which the anviels can produce by their expulsion of as much of their
but particularly in the want of blood enough in the brain. All the capillaries having too little arterial blood reaching them, being overloaded with venous blood, the stagnation of fullness in the veins, indicated by the lividity. This gasping respiration continues, crisis & jactitations increase, lividity of face becomes more marked, the eyes brighter & wilder. Such a picture must correspond with the expression — help — clearer than words could convey it. This condition continues, may increase, soon to be followed by a growing decline in all the urgency of all the symptoms. The crisis & jactitations are less frequent, the depression less urgent, but interspersed by occasional exacerbation,
the eye becomes dull and heavy, the patient dozy. The pulse pulseable, flabby, indicative of drowsiness gives place to sleep which is unnatural therapy. The pulse now gradually disappears, the stethoscope still reveals the condition of the heart which beating at from about 140 to 170, slows abruptly, growing incomplete gives one the idea when listening of a struggle which cannot be long maintained.

The respirations become slower slower, the cardiac action, more or less less less complete of the scene closes in calm, having already spoken of the injury to the cardiac action.
of the presence of a large quantity of fluid in the pericardium, let us consider the cause of its retention there. Some which actually tend to its increase. As long as the inflammation continues, the exudation increases. But here it is difficult to find where inflammation exists (the other causes of increase of fluid or prevention of its absorption being). We have the lymphatic coating the pericardial surface to a greater or less extent. This lessens, according to its amount, the surface which, when the inflammation subsides, is the only absorbing surface for the removal of the contained pericardial fluid. Also of a considerable extent.
Of a large amount of effusion
is poured out before the inflam-
atation ceases, this actually assists
in preventing its own absorption,
by its weight pressing on the
capillaries, hindering or preventing
absorption by them. Now if the
fluid thus injuriously press on
the capillaries & prevent their ab-
sorptive action, this mechanism
turns what ought to be an ab-
sorbing membrane into a secreting
one, for a certain amount of
pressure still exists in the arteries
supplying these capillaries. If this
pressure continued must cause
effusion from the capillaries into
the cavity of the pericardium. True
the amount so poured out must
be small, but though small in
Amount it will simply increase by its account the impediment here referred to. The presence of a large quantity of fluid as already mentioned will press on the heart itself to injure its function by interfering with its action, and also produces stretching of the sac (mentioned before). This stretching is reduced during contraction of the ventricles by the bulk of blood propelled onward by the heart (that bulk as before explained being less than normal on account of the pressure outside the heart). But what of the null action of the heart—dilatation? This act has not been performed again.
the fluid to be displaced. The pericardium is to be stretched.
This as before explained is in a direction in which the heart has very little power. These two things produce a hindrance in the circulation at its centre which increases venous stagnation which is evidently one very efficient obstacle to the effused fluid being absorbed. In fact were there no other element in the way, venous congestion would of itself retard or according to its amount prevent absorption of fluid anywhere. Hence we will be readily allowed we must remove venous congestion to allow of absorption. I remove extra-cardiac pressure, which is
the great cause of venous congestion, or what I will pre-

vent in treatment, prevent venous congestion by preventing its cause, namely, pressure of peri-

cardial fluid limiting the filling of the cavities of the heart.

In such a case the indications are, to prevent pressure on the heart from peri-cardiac effusion, or remove it if injurious to its amount; prevent venous congestion, its consequences, or relieve it if already present.

The venous congestion as will be gathered from the foregoing is the direct result of the pressure of the peri-cardial fluid. The former when sufficiently de-

veloped tends to increase the quantity
of the latter, I certainly prevent its removal in the only way in which that is possible by nature—absorption. The rapid removal of pericardial fluid is a thing that we should always avoid, because of the possible fatal result. There is too much blood behind the cardiac cavity in the distended pulmonary & systemic vessels, & if the intra-cardial pressure is removed, especially by such an instrument as a trocar & cannula of some size, or by quick abstraction with the exhausting syringe or aspirator, the sudden withdrawal of sectional pressure allows the distended vessels (pulmonary & systemic) to force their contents forward into the weakened & partly exhausted aorta, & ventricle; the consequence
Contents as can be produced by their now pulsile force into the ventricles, on whose walls both fluid & stretched as now act, gradually their entire bulk in diastole, the most important of all limiting the capacity of all the cavities. The consequence of this partial reflection of the cavities, is that they have too little blood to contract upon. This stunts the whole heart, its rhythm is broken, & irregularity results. Too little blood in consequence reaches the arterial system. From this arises the urgent dyspnea which is the patient's chief, sometimes the only distress complained of. The breathing is quite different.
from the former briefly rapid breathing. It is gasping, irregular, spasmodic, long gasps or deep sighs, followed by quicker breathing then sighs again. Quite different from the hurried breathing due to bronchial diseases. In this Cardiac dyspnea there is no impediment to entrance or exit of air, but still there is a distress more urgent than in any chest affection. It is the pulmonary circulation which is deranged, both by too little blood reaching it from the compressed right side and too little reaching the left side, too little blood therefore in the arterial system. These grunts or sighs and jactitations being the expression both of the want in the Lungs...
right one from its lower position. Against this external pressure on the ventricular wall, the auricular pressure contracts its power is insufficient. The ventricular wall itself is unadapted, from its hollow nature, to resist force applied to its external surface & pressing towards its cavity, even supposing it had a certain amount of suction power which some physiologists would give it. Pressure by the mere presence of fluid in the pericardium will be exerted on all the cavities, but speaking especially of the ventricle at present, the right ventricle will be more hindered in its action than the left on account of the pubic resistance which its
thinner walls offer to the external pressure. These cavities therefore don't get sufficiently filled with blood; the auricles, becoming gradually less able to displace the fluid pressing on the outside of the heart, the ventricular walls sag—nation begins at the auricles, & affects all the parts behind these; but the ventricles, being insufficiently filled, their time of contraction [is] nevertheless being arrested, the contract as it were with adrenaline. This muscle or it may be extra force sustained on a less quantity of blood than these cavities ought to contain, expel their contents violently forward, but as the force is not, as normally, graduated to the requirements
A few words may here be offered on what I consider the cause of this and other forms of irregular cardiac action. With fever, I have undoubtedly to do with increased rapidity of heart's action, but not I should say with the irregularity until the heart becomes somewhat irritable. Hence, it may form one element in producing irregularity. But in the beginning of such a case as this, I am inclined to believe in its production by another cause. The presence of fluid, in quantity, is an abnormal condition in the pericardial sac. This hides the heart in its dilatations, and, by its presence,
When a considerable quantity by its pressure also; not inter-
fering directly with the cardiac contractions, but at the same
time preventing, to the proper extent, one all-important condition on
which the due performance of that act depends—namely the
proper filling of the cavities of the heart. The proper filling of the cav-
ities is prevented by the fluid in the pericardium pressing upon
the walls of these cavities, preventing their full dilatation. Reflection with
blood, by the auricles, propelling their contents onwards into the
ventricles, too. Causing their dilatation. The auricles are also pressed
upon when there is a large quan-
tity of fluid, especially the right
of which will be immediate engorgement & destruction of these cavities, with the result of an immediate block in the circulation, the pulse heart not being able to clear itself of such a sudden influx of blood, sudden death resulting from cessation of the heart.

If one should meet with such a case in which there was a large pericardial effusion & venous congestion & if it be deemed necessary to tap, it would be wise, as a preliminary to that operation to abstract blood from the jugular vein, in order to prevent as much as possible the too sudden influx of blood into the heart during the operation, its probable result.
True this would only reach the systemic venous circulation directly, but would certainly affect the pulmonary system secondarily, if a little time elapsed before the performance of paracentesis pericardii.

Now of the preventive treatment which has been more than hinted. How are we to prevent pressure on the heart from pericardial fluid, or venous systemic thoracic congestion? It will be seen from the frequent mention of the intimate relation of these two conditions to each other, that if we can prevent extra-cardiac pressure by fluid we prevent the great cause of these congestions, therefore preventive treatment resolves itself into dealing with the pericardial fluid,
either preventing its effusion, or causing its removal before injury results from its presence. What I propose therefore after the usual remedies have failed to stop the increase of fluid in the peri-cardia-in, whose symptoms (low slight) indicate that injury to the circulation has begun, from that fluid, especially when such symptoms are rapidly produced, I increase as in the case given, I think we will be able to prevent injury by the direct removal of the fluid as it is effused, not waiting till it accumulates so as to interfere. Supplying with the cardiac action, but drawing it off repeatedly as the fluid that it increases of that symptoms indicate its increase, just the same as we
would prescribe an emetic for a child with bronchitis, which was too young to expectorate when we found that mucus had accumulated to an extent to cause restlessness and increased difficulty of breathing, especially by making out the actual presence of a quantity of mucus in the chest by auscultation.

The method (for it is so mild that it hardly deserves to be designated with the term of operation) of the repeated removal of small quantities of fluid from the pericardium which I would advise is by a very fine trocar attached to a small syringe. With this draw off a small quantity of fluid, an ounce or two
volumes at a time. An aspirator of the same capacity, with fine needle, may be used. In either case we must draw off the fluid slowly, so as to avoid too sudden alteration of pressure which might produce pain. In I need hardly add that whatever instruments are used in this modified punctation, they ought to be perfectly clean. The patient ought not to be deprived of the additional security which antisepctic precautions would give in its performance. Frequent slight tapings with fine trochar could not injure the thoracic wall over the peri-cardium.
The sudden diminution of the heart, within the pericardium, and the corresponding increase in the Cardiac Cavities, by the blood being pressed thither from the engorged & dilated vessels behind these cavities, produces a too sudden overfilling of the heart in all its cavities; the weakened organ cannot resist this sudden influx & cessation of the heart's action results. In this way I think is explained the fact that results from circumcisions performed too late.

From the mechanism which has now I hope been explained with sufficient clearness, this premonition
Treatment,—the removal of small quantities, at a time, of pericardial fluid, which although at present theoretical seems to be so evident that it is hoped it will prove as useful in practice as it looks in theory.

Many points in this paper might have been dilated on at length, but the important ones seem to have been sufficiently dealt with for the purpose in hand.

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