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

The subject of the case was a married woman, age 35. The first acquaintance with the patient was 10 months previously, during confinement, which from a malposition of the child's head at the brim 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 occurence in the history, the subject being previously healthy, & was so from that time to the time of her present attack.

June 15. 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 patients condition. There was evid-ent distress,- increased thirst, lips parched, distressed breathing (not like an ordinary dyspnoea). 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 pre-vented prolonged examination; the percussion dulness was ex-tensive although no exact measurements 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, jactitations considerable, orthopnœa constant, continual restlessness. This night no 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 pallid livid hue than lips. Heart was now too rapid, -130 per minute- to distinguish more than its normal sounds which were indistinct, & 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 interrupted by intervals of calm. This & the growing lividity of the face

Hips indicated that the end was near. Pulse like the heart very rapid, irregular & becoming feeble. The condition of the cranial circulation being indicated by the mental distress jactitation, & breathing. Wandering & delerium were interrupted by occasional lucid intervals which became shorter & less frequent as the case advanced.

Evening visit,— Signs & symptoms aggravated.

24th Morning. The night had been worse than any previous, she having roused considerably from the previous days semi-

-comatose

condition. Her screaming was so loud as to be heard for some distance in the neighbourhood. 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 & rapidity of the breathing it was somewhat difficult to understand her interrupted articulation.

Heart now very irregular. Pulse about 140–150 being so feeble & irregular that the exact number was uncertain. When properly roused, the wildness of the gaze was, if possible,

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, & seemed to fall into whatever position the surroundings 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 <u>cardiac bearings</u>.

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 draw off the fluid with trochar & canula. What deterred me from this was the knowledge that patients are said to have died in the physicians 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 practicing it.

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

The early occurrence & the rapid increase of the distress of breathing the large area of dulness on percussion, the rapidity, irregularity & tumultuous action of the heart all indicated the abrupt interference of the pericardial effusion with both Circulation & respiration.

The irregularity was characterised by one large & powerful contraction of the heart beating very strongly against the chest-wall, followed by contractions growing less & less powerful & more rapid, till the 6th or 7th was reached; then came a long interval which ended, & 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, & became more feeble, when neither the long interval, large beat, nor its followers could be so well made out, but there was perceptable irregular irregularity of both heart & 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 & powerful contraction). There is no resistance ~~to the~~ until the ventricular wall becomes tense on the small quantity of fluid in it, & now it expels this onward. This shock or surprise is very quickly followed by the other 6 or 7 more rapid, less powerful contractions, which are quicker & less ~~powerful~~ effectual from two causes. First, the prior shock & the second less quantity of blood in the ventricle to be propelled, as there is really no time given

& refill the ventricles. Examples of similar kinds of surprise & shock are seen in manual labour & machinery; as in working an ordinary pump. When one is depressing & raising the handle vigorously, 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 got accustomed to, & this time prepare & expend the usual amount of force. Meeting with very little resistance the handle goes down with a bang, & we accordingly experience a surprise & receive a shock proportioned

& the amount of force expended.
This corresponds & the large &
violent contraction of the ventricle;
but we knowing that something
is wrong dont repeat the ex-
-periment until we enquire
into & rectify matter. The heart
cant do so, but more & more ineff-
-ectual contractions follow this
shock until the heart as it were
gets exhausted, & then follows the
long pause, & be again followed
by the same shock & rapid con-
-tractions. We have perhaps a
nearer resemblance to the case
of the heart in a piece of mach-
-inery. Take a steam engine
working up to the required power,
& driving ~~up to the required~~
a threshing mill.

when the mill is equally fed with the spread-out sheaves of grain, the engine works quite steadily, but when from any cause the feeding flags, immediately the engine acts faster & faster, as there is less to do, in fact as the machine becomes emptier; then again it is properly fed & the whole goes on steadily until the same cause brings 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 a Cardiac irregularity which admits of the same explanation. Here

unlike the half-filled ventricle occasioned by the pressure of the pericardial fluid, we have a ventricle quite full in Mitral dilatation & nearly so in Stenosis unless the contraction of that orifice be very great. We have here plenty of blood & be propelled but the mechanism for its being expelled from the ventricle in the <u>onward direction only</u> is disordered. The ventricle contracts on its contents & expells them 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 filled ventricle in consequence. Here, in the

Mitral case, one part of the 2nd
blood, proportioned to the incom-
-petence, 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) resisting 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 ventricle empties

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 & amount of the valvular lesion & the work or 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 infiltrement & the ventricular ~~action~~ dilatation. When the heart is surrounded by the fluid, its action is hindered not

only by the quantity of 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 & stretch the membrane. Now what is the effect of this on the contained 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 & the utmost which the auricles can produce by their expulsion of as much of their

but particularly 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 & fullness
in the veins, indicated by the
lividity. This gasping respiration
continues, cries & jactitations
increase, lividity of face becomes
more marked, the eyes brighter
& wilder. Such a picture as my gaze with
the expression — help — clearer than
words could convey it. This con-
-dition continues & may increase
soon & be followed by a growing
decline in ~~all~~ the urgency of
all the symptoms. The cries &
jactitations are less frequent, the
dyspnœa less urgent, but inter-
-upted by occasional exacerbations

the eye becomes dull & heavy, the patient drowsy. The pulse feeble & flattening. Lividity increases drowsyness gives place to sleep which is unnatural & heavy. The pulse now gradually disappears, the stethoscope still reveals the condition of the heart which beating at from about 140 to 170, sounds abrupt, & 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 & more~~ less & less complete & the scene closes in coma.

Having already spoken of the injury to the cardiac action

of the presence of a large quantity of fluid in the pericard-
-ium, let us consider the causes 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 ceases & the other causes of increase of fluid or prevention of its absorption begin.
We have the lymph coating the pericardial surface to a greater or less extent. This lessens according to its amount, the surface which, when the inflam-
-ation subsides is the only absorb-
-ing surface for the removal of the contained pericardial fluid.
Also if a considerable & especially

if a large amount of effusion is poured out before the inflammation ceases, this actually assists in preventing its own absorption, by its weight pressing on the capillaries, & limiting or preventing absorption by them. Now if the fluid thus injuriously press on the capillaries & prevent their absorptive action, this mechanism turns what ought to be an absorbing membrane into a secreting one, for a certain amount of pressure still exists in the arteries supplying these capillaries & 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 amount the impediment here referred to.

The presence of a large quantity of fluid as already mentioned will press on the heart itself & impair its ~~action~~ function by interfering with its action, & 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 next action of the heart - dilatation? This act has to be performed again

the fluid to be displaced & the pericardium to be stretched. This as before explained is a direction in which the heart has very little power. These two things produce a hinderance 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 as will be readily allowed we must remove venous congestion to allow of absorption, & remove extra-cardiac pressure, which is

the great cause of venous congestion, or what I will propose in treatment, prevent venous congestion by preventing its cause, namely, pressure of pericardial fluid limiting the filling of the cavities of the heart.

In such a case the indications are, — to prevent pressure on the heart from pericardiac effusion, or remove it if injurious by 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 pericardial fluid, & the former when sufficiently developed tends to increase the quantity

of the latter, & certainly prevents 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 & even probable, quickly fatal result. There is too much blood behind the cardiac cavities in the distended pulmonary & systemic vessels, & if the extra-cardial pressure is removed, especially by such an instrument as a trocar & canula of some size, or by quick abstraction with the exhausting syringe or aspirator, the sudden withdrawal of external pressure allows the distended vessels (pulmonary & systemic) to press their contents forward into the weakened & partly exhausted auricles & ventricles, the consequence

Contents as can be propelled by their now feeble force into the ventricle, on whose walls both fluid & stretched sacs now act, gradually lessening their entire bulk in diastole, & most important of all limiting the capacity of all the cavities. The consequence of this partial repletion of the cavities, is that they have too little blood to contract upon. This stuns the whole heart, its rythm is broken, & irregularity results. Too little blood in consequence reaches the arterial system. From this arises the urgent dyspnœa which is the patient's great, & sometimes the only distress complained of.

The breathing is quite different

from the former simply 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 dyspnoea 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 & by too little reaching the left side, too little blood therefore in the arterial system. These gasps & cries & jactitations being the expression both of the want in the Lungs

right one from its lower position. Against this external pressure on the ventricular walls the auricular ~~pressure~~ contractile power is insufficient. The ventricular wall itself is unsuited, from its hollow nature, to resist force applied to its external surface & pressing towards ~~the~~ 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 the walls of all the cavities, but speaking specially of the ventricles at present; the right ventricle will be more hindered in its action than the left on account of the feebler resistance which its

thinner walls offer to the external pressure. These cavities therefore dont get sufficiently filled with blood; the auricles becoming gradually less able to displace the fluid pressing on the outside of the the Ventricular walls, stagnation begins in the auricles & affects all the parts behind these; but the Ventricles being insufficiently filled their time of contraction nevertheless having arrived, they contract as it were with impatience, & this usual or it may be extra force expended on a less quantity of blood than these ~~vessels~~ 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 & some other forms of irregular cardiac action.
High fever has undoubtedly to do with increased rapidity of hearts action, but not I should say with the irregularity until the heart becomes somewhat inflamed & therefore irritable, then 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 hinders the heart in its dilatations & rests, by its presence,

of urine in considerable quantity by its pressure also; not interfering 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 ventricles is prevented by the fluid in the pericardium pressing upon the walls of these cavities, preventing their full dilatation & repletion with blood, by the auricles propelling their contents onwards into the ventricles & so causing their dilatation. The auricles are also pressed upon when there is a large quantity of fluid, especially the right

of which will be immediate
engorgement & distention 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 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 con-
-gestion & 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, & venous systemic & pulmonary 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-cardial 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 pericardium, & when symptoms
(even slight) indicate that injury
to the circulation has begun, from
that fluid, & especially when such
symptoms are rapidly produced
& increase as in the case given,
I think we will be able to prevent
~~death~~ injury by the direct removal
of the fluid as it is effused, not
waiting till it accumulates so as
to interfere seriously with the
cardiac action, but drawing it
off repeatedly as we find that it
increases or 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 & 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 dignified
with the name of operation) of the
repeated removal of small quan-
-tities 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 quantities
of fluid, an ounce or two

ounces at a time. An aspirator of the same capacity, with fine needle, may be used. In ~~both~~ either case we must draw off the fluid slowly, so as to avoid too sudden alteration of pressure which might produce fainting. I need hardly add that whatever instruments are used in this modified paracentesis, ~~they~~ ought to be perfectly clean & the patient ought not to be deprived of the additional security which antiseptic precautions would give in its performance. Frequent slight tappings with fine trocar could not injure the thoracic wall over the peri-cardium.

I may repeat that—
The too sudden diminution of 39.
pressure within the pericardium & 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 cant uphill this sudden influx & cessation of the hearts action results. In this way I think is explained the fatal results from paracentesis performed too late.

From the mechanism which has now I hope been explained with sufficient clearness, this prevention

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 plain in theory.

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

Alexander Henry M.B.

10 W. Newington Terrace

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